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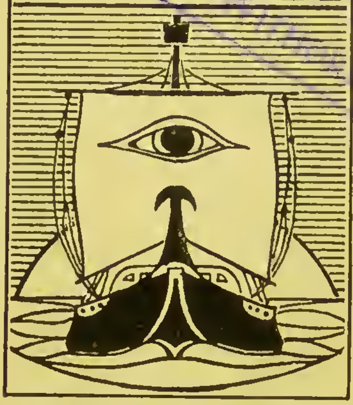

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ROYAL SOCIETY.

# REPORTS

OF THE

# SLEEPING SICKNESS COMMISSION.

## No. I.

1. Presence of Trypanosoma in Sleeping Sickness. By Dr. Aldo Castellani.
2. Progress Report on Sleeping Sickness in Uganda. By Lieut.-Col. David Bruce, F.R.S., R.A.M.C., and Dr. David Nabarro.

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VIEW OF LABORATORY, ENTEBBE. [*Frontispiece.*

# 1.

---

# PRESENCE OF TRYPANOSOMA IN SLEEPING SICKNESS.

By ALDO CASTELLANI, M.D.

[Dated Entebbe, Uganda, April 5, 1903.—Received May 8, 1903.*]

On the 12th November, 1902, when examining a specimen of cerebro-spinal fluid taken by lumbar puncture during life from a well-marked case of *sleeping sickness*, I was surprised to observe a living Trypanosoma. Since that date I have made as many observations in this direction as possible, and the results are to my mind sufficiently surprising to excuse me for presenting this preliminary note.

These trypanosomes are not in large numbers, so that to find them it is necessary to draw off at least 15 c.cs. of the cerebro-spinal fluid. It is better to reject the first few c.cs. as they are apt to contain blood. When the fluid comes away clear, 10 c.cs. are collected and centrifuged for 15 minutes. At the end of this time there is found at the bottom of the tube a slight deposit of whitish sediment, and in some cases also a minute trace of blood.

The liquid above the sediment is poured off and the sediment examined under a moderately low power of the microscope. As the trypanosomes are at first fairly active they are easily detected.

The following tables represent the results of this investigation :—

---

* This has already been published in the 'Proceedings of the Royal Society,' vol. 71, 1903, p. 501.

Table I.—Sleeping Sickness Cases.

| No. | Name. | Sex. | Age. | Date. | Stage of disease. | Microscopic appearance of sediment. | Presence of trypanosoma in cerebro-spinal fluid. | Remarks. |
|---|---|---|---|---|---|---|---|---|
| 1 | Mundo .......... | M. | 15 | 12/11/02 | 3rd | A few leucocytes, the majority of which are mononuclear. Some very rare red blood corpuscles. | Present | |
| 2 | Maoli.... ....... | M. | 18 | 25/11/02 | 3rd | Some few leucocytes and R.B.C. | Absent | |
| 3 | Aritzo ........... | M. | 25 | 7/12/02 | 3rd | Some leucocytes. No R.B.C. | Absent | |
| 4 | Manika .......... | F. | 10 | 15/12/02 | 3rd | A few leucocytes. Very few R.B.C. | Present | Patient died on December 18, 1902. No complications. In fluid from lat. vent. Tryp. present. |
| 5 | Ialika ........... | F. | 22 | 15/12/02 | 2nd | A few leucocytes. Very few R.B.C. | Absent | |
| 6 | Asmeni .......... | F. | 8 | 15/12/02 | 3rd | Some leucocytes and many R.B.C. | Absent | |
| 7 | Bolenti .......... | M. | 10 | 22/12/02 | 3rd | Some leucocytes. No R.B.C. | Present | In fresh preparations of blood taken from a finger the same day I found a few trypanosomes apparently similar to those found in the lumbar puncture liquid, only their movements were apparently more lively. |
| 8 | A.............. | M. | 20 | 5/1/03 | 3rd | Few leucocytes and some R.B.C. | Absent | |
| | | | | 7/1/03 | .. | Some leucocytes and very few R.B.C. | Absent | |

Table 1.—Sleeping Sickness Cases—*continued.*

| No. | Name. | Sex. | Age. | Date. | Stage of disease. | Microscopic appearance of sediment. | Presence of trypanosoma in cerebro-spinal fluid. | Remarks. |
|---|---|---|---|---|---|---|---|---|
| 9 | Makassa ......... | F. | 25 | 25/1/03 | 3rd | Some leucocytes. No R.B.C. | Present | |
| 10 | Kaperi II......... | M. | 14 | 25/1/03 | 2nd | A few leucocytes and very few R.B.C. | Absent | |
| 11 | Ally II ......... | M. | 30 | 2/2/03 | 3rd | Some leucocytes and R.B.C. | Absent | |
| 12 | Moereza ......... | M. | 30 | 10/2/03 | 2nd | A few leucocytes and R.B.C. | Absent | |
| 13 | Budara ......... | M. | 22 | 27/2/03 | 2nd | Some leucocytes and very few R.B.C. | Present | |
| | | | | 2/3/03 | .. | Some leucocytes and very few R.B.C. | Present | |
| 14 | Nombi ......... | F. | 30 | 27/2/03 | 1st | Few leucocytes and R.B.C. | Absent | |
| | | | | 24/3/03 | 2nd | Some leucocytes and R.B.C. | Absent | |
| 15 | Fatoma ......... | F. | 18 | 27/2/03 | 2nd | Very scarce leucocytes and no R.B.C. | Absent | |
| | | | | 4/3/03 | .. | Few leucocytes and R.B.C. | Absent | |
| | | | | 26/3/03 | .. | Few leucocytes and R.B.C. | Absent | |
| | | | | 1/4/03 | .. | Some leucocytes and a few R.B.C. | Present | |
| 16 | Zenabu ......... | F. | 22 | 24/3/03 | 1st | No R.B.C. .. .. | Absent | |
| | | | | 28/3/03 | 1st | Some R.B.C. .. .. | Absent | |
| | | | | 1/4/03 | 1st | Some R.B.C. .. .. | Absent | |

Table I.—Sleeping Sickness Cases—*continued.*

| No. | Name. | Sex. | Age. | Date. | Stage of disease. | Microscopic appearance of sediment. | Presence of trypanosoma in cerebro-spinal fluid. | Remarks. |
|---|---|---|---|---|---|---|---|---|
| 17 | Benjamin | M. | 20 | 25/3/03 | 2nd | .. | Absent | |
| | | | | 28/3/03 | .. | Some few leucocytes and R.B.C. | Absent | |
| | | | | 29/3/03 | .. | Some few leucocytes and R.B.C. | Absent | |
| 18 | Zakibu | M. | 25 | 25/3/03 | 2nd | Few leucocytes and R.B.C. | Present | |
| | | | | 27/3/03 | 2nd | Few leucocytes and R.B.C. | Present | |
| 19 | Seera | M. | 25 | 26/3/03 | 2nd | .. | Present | |
| 20 | Kimbra | M. | 55 | 26/3/03 | 3rd | Some leucocytes. No R.B.C. | Present | |
| 21 | Abdulla | M. | .. | 26/3/03 | .. | No R.B.C. .. .. | Present | |
| 22 | Kagoya | F. | 20 | 26/3/03 | 3rd | No R.B.C. .. .. | Present | |
| 23 | Keogaffum | M. | 55 | 27/3/03 | 2nd | Some leucocytes. No R.B.C. | Absent | |
| 24 | Jaeobo | M. | 20 | 1/4/03 | | .. | Absent | |
| | | | | 28/3/03 | 3rd | Some leucocytes. No R.B.C. | Present | The trypanosomes were much more numerous than in other cases. |
| 25 | Iegobaza | M. | 40 | 27/3/03 | 2nd | Few leucocytes. No R.B.C. | Present | |
| 26 | Ibsarara | F. | 35 | 27/3/03 | 3rd | Some leucocytes. No R.B.C. | Present | |
| 27 | Leobeni | M. | 25 | 28/3/03 | 2nd | No R.B.C. .. .. | Present | |
| 28 | Kidorme | M. | 20 | 28/3/03 | 2nd | Some leucocytes and R.B.C. | Present | |

Table I.—Sleeping Sickness Cases—*continued.*

| No. | Name. | Sex. | Age. | Date. | Stage of disease. | Microscopic appearance of sediment. | Presence of trypanosoma in cerebro-spinal fluid. | Remarks. |
|---|---|---|---|---|---|---|---|---|
| 29 | Keagabidoia...... | M. | 55 | 28/3/03 | 3rd | Some leucocytes and R.B.C. | Absent | |
| | | | | 1/4/03 | 3rd | Some leucocytes and R.B.C. | Absent | |
| 30 | Kitaroma........ | M. | 25 | 28/3/03 | 2nd | Some leucocytes and R.B.C. | Absent | Only 5 c.c. of liquid collected. |
| | | | | 2/4/03 | 2nd | Some leucocytes and R.B.C. | Absent | |
| 31 | Waiswa ........ | M. | 10 | 29/3/03 | 2nd | Some leucocytes and R.B.C. | Present | |
| 32 | Kaperi I. ........ | M. | 8 | 23/3/03 | 3rd | No R.B.C. .. .. | Present | Trypanosoma present also in the fluid taken from lateral vent. at the *post mortem.* |
| 33 | Matasa ........ | M. | 28 | 29/3/03 | 2nd | Few leucocytes and R.B.C. | Absent | |
| | | | | 2/4/03 | 2nd | Few leucocytes and R.B.C. | Absent | |
| 34 | Decodeno........ | M. | 25 | 31/3/03 | 2nd | Few leucocytes and R.B.C. | Present | |

Table II.—Controls.

| No. | Name. | Sex. | Age. | Date. | Disease. | Microscopic appearance of sediment. | Presence of trypanosoma. | Remarks. |
|---|---|---|---|---|---|---|---|---|
| 1 | Doanira ......... | M. | 35 | 11/1/03 | Chronic nephritis | Very few leucocytes and R.B.C. | Absent | |
| 2 | Kano Buringo.... | M. | 30 | 24/3/03 | Trypanosoma fever | Very few leucocytes and R.B.C. | Absent | Dr. Baker found *Trypanosoma Gambiense* in blood some days ago. |
| 3 | Landu ......... | M. | .. | 24/3/03 | Enlargement of femoral glands | Few leucocytes. No R.B.C. | Absent | |
| 4 | Kamsuro ........ | M. | .. | 28/3/03 | Trypanosoma fever | Few leucocytes and R.B.C. | Absent | Dr. Baker found *Trypanosoma Gambiense* in blood the same morning. |
| 5 | Zake ............ | M. | 25 | 30/3/03 | Cellulitis | Few leucocytes. No R.B.C. | Absent | |
| 6 | Scongo .......... | .. | .. | 30/3/03 | Itch | Very few leucocytes and R.B.C. | Absent | |
| 7 | Pio.............. | M. | 12 | 30/3/03 | .. | Very few leucocytes and R.B.C. | Absent | |
| 8 | Kaperi III ...... | M. | 25 | 30/3/03 | Itch | Very few leucocytes and R.B.C. | Absent | |
| 9 | Eliza............ | F. | 18 | 30/3/03 | Yaws | Very few leucocytes and R.B.C. | Absent | |
| 10 | Bofralour........ | M. | 16 | 31/3/03 | Pleuritis | Very few leucocytes and R.B.C. | Absent | |
| 11 | Zanabu II........ | F. | 30 | 31/3/03 | Headache | Very few leucocytes and R.B.C. | Absent | |
| 12 | Jordien Murjan .. | M. | 35 | 31/3/03 | Trypanosoma fever | Very few leucocytes and R.B.C. | Absent | Dr. Baker found *Trypanosoma Gambiense* in blood the same morning. |

Table I shows that in 34 cases of sleeping sickness, the trypanosomes were found in the cerebro-spinal fluid taken by lumbar puncture during life in 20 cases, giving a rate of 70 per cent.

On two occasions I also examined in the same way fluid from the lateral ventricles and in both cases found the same parasite. In blood I found the trypanosoma once with certainty.

It may be thought that the trypanosomes are found in the cerebro-spinal fluid on account of the trace of blood which sometimes forms part of the sediment. But it will be seen from the table that in several cases there was no trace of blood.

Table II shows that in 12 cases of ordinary disease, the cerebro-spinal fluid taken during life by lumbar puncture, in no case contained trypanosoma, and it is important to note that 3 of these controls were cases of the usual trypanosoma fever, as described by Forde and Dutton, Manson and Daniels.

Here it may be remarked that trypanosoma fever is by no means uncommon among the natives in Uganda, 3 cases having been met with by Dr. Baker, one of the colonial surgeons here (Entebbe), within the last 3 weeks. I understand that Dr. Baker is publishing this most interesting observation. It must be clearly understood that these cases of trypanosoma fever bear no resemblance in their clinical features to sleeping sickness.

The trypanosoma found in the cerebro-spinal fluid of sleeping sickness does not as far as I have been able to make out differ materially in size and shape from the species one finds in the blood of trypanosoma fever, *Trypanosoma Gambiense* (Dutton), but possibly it is to be differentiated from this one, because in it, as a rule, the micro-nucleus lies nearer the extremity and the vacuole is apparently larger. Besides, its movements are not apparently so active, but this fact might be due to the effects of the centrifuge.* In case it should prove to be a new species, the trypanosoma I have described might be called from the country where I have found it first—*Trypanosoma Ugandense.*

## *Relation of the Trypanosoma to Sleeping Sickness.*

At the *post-mortem* examination of 80 per cent. of the cases where I found during life the trypanosoma, I grew from the blood of the heart and from the liquid of the lateral ventricles the variety of streptococcus I described many months ago in my first note. Up to that time I had never found the trypanosoma, but this is easily explained by the fact that I did not use the technique I have described in this note, viz., examination of a *large* quantity of liquid after long use of the centrifuge.

---

* It is interesting to note also that the trypanosoma found in sleeping sickness moves always with the so-called posterior extremity in front, whereas *T. Gambiense*, according to Dutton, proceeds usually with the anterior extremity (flagellum) in front.—*July* 29, 1903.

Influenced by my last investigations I would suggest as a working hypothesis on which to base further investigation that sleeping sickness is due to the species of trypanosoma I have found in the cerebro-spinal fluid of the patients in this disease, and that at least in the last stages there is a concomitant streptococcus infection which plays a certain part in the course of the disease.

[*August*, 1903.—The Trypanosome described in this communication has, I find, been named *Trypanosoma Castellanii* by Kruse. See "Ueber das *Trypanosoma Castellanii*, den Erreger der Schlafkrankheit der Neger."—'Sitzungsberichte der Niederrhein. Gesellsch. f. Natur-u. Heilkunde zu Bonn, 18 Mai, 1903.']

## 2.

# PROGRESS REPORT ON SLEEPING SICKNESS IN UGANDA.

By Lieut.-Col. DAVID BRUCE, F.R.S., R.A.M.C.,

AND

DAVID NABARRO, M.D.

(*SLEEPING SICKNESS COMMISSION.*)

[Dated Entebbe, Uganda, May 29.—Received June 29, 1903.]

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The Commission arrived in Entebbe, Uganda, on March 16, 1903, and were met by Dr. Castellani, a member of the Sleeping Sickness Commission, sent out by the Royal Society in May, 1902.

Dr. Castellani informed us of the work he had done, one especially interesting observation being that he had discovered trypanosomes in the cerebro-spinal fluid in five out of fifteen cases of sleeping sickness. Dr. Castellani remained in Entebbe for three weeks after the arrival of the new Commission, and during this time he examined twenty-nine further cases for trypanosomes, with the result that 70 per cent. were found to contain these parasites. Dr. Castellani we presume has already published these results.* After his departure the Commission continued to pursue this line of work.

### 1. *Examination of the Cerebro-spinal Fluid of Cases of Sleeping Sickness for Trypanosomes.*

The method employed is the same as that employed by Dr. Castellani. 10 c.c. of cerebro-spinal fluid was taken by lumbar puncture. The fluid is centrifuged for a quarter of an hour, and the whole of it poured away except the little which clings to the bottom and sides of the tube. The sediment is stirred up in this and examined under a low power, 150 to 200 diameters of the microscope; Zeiss 16 mm. apochromatic objective and Nos. 8 or 12 eyepiece do well. The trypanosomes are never very numerous. In some cases one is found only after looking through several slides, in others two or three may be seen in one field.

The cerebro-spinal fluid in Sleeping Sickness cases differs slightly from that found in healthy persons. It usually has a very slight tinge of yellow due to the presence of a few red blood corpuscles, and also has more cellular elements in suspension. These cellular elements are mostly lymphocytes.

The healthy cerebro-spinal fluid on the other hand is as clear and limpid as distilled water and contains no sediment.

It may be assumed from Table 1 that every case of sleeping sickness has trypanosomes in the cerebro-spinal fluid, even in the early stages of the disease. It must be borne in mind that, as a rule, they are in small numbers, and that one examination will not always decide their presence or absence; success is very much a matter of care and patience.

---

* [Dr. Castellani's results are published in the preceding Report (1). From this it will be seen that Dr. Castellani had ascertained with certainty the presence of trypanosoma in the blood of one case; in another case the result of the observation was not certain. Dr. Castellani had also observed developmental forms of the parasite. See 4 (in the press).—Sec. R.S.]

Table I.—The Result of this Procedure in Forty Cases.

| Date. | Name. | Sex. | Age. | Duration of case. | No. of examination. | Trypanosoma. |
|---|---|---|---|---|---|---|
| 1903. | | | | | | |
| May 14 | Kaperi | Male | 8 | 3rd stage | 1 | Present. |
| Mar. 26 | Seera | ,, | 25 | 1st ,, | 1 | ,, |
| ,, 26 | Budara | ,, | 22 | 2nd ,, | 1 | ,, |
| ,, 26 | Kimbra | ,, | 30 | 2nd ,, | 1 | ,, |
| ,, 26 | Kagoya | Female | 20 | 3rd ,, | 1 | ,, |
| ,, 27 | Zeboganza | Male | 40 | 1st ,, | 2 | ,, |
| ,, 27 | Yakubu | ,, | 12 | 2nd ,, | 1 | ,, |
| ,, 28 | Kidorme | ,, | 20 | 2nd ,, | 1 | ,, |
| ,, 28 | Leobeni | ,, | 25 | 3rd ,, | 1 | ,, |
| ,, 29 | Waiswa | ,, | 10 | 1st ,, | 1 | ,, |
| ,, 31 | Dekodemo | ,, | 25 | 3rd ,, | 1 | ,, |
| Apr. 1 | Fatoma | Female | 18 | 1st ,, | 2 | ,, |
| ,, 6 | Katola | Male | 25 | 1st ,, | 1 | ,, |
| ,, 6 | Esaka | ,, | 28 | 1st ,, | 1 | ,, |
| ,, 6 | Nakaiba | Female | 10 | 1st ,, | 1 | ,, |
| ,, 6 | Zakibu | Male | 20 | 2nd ,, | 1 | ,, |
| ,, 6 | Warosansa | ,, | 32 | 2nd ,, | 1 | ,, |
| ,, 8 | Jansi | ,, | 25 | 1st ,, | 1 | ,, |
| ,, 9 | Feragi | ,, | 12 | 1st ,, | 1 | ,, |
| ,, 10 | Katoola | ,, | 20 | 1st ,, | 1 | ,, |
| ,, 10 | Donah | ,, | 38 | 1st ,, | 1 | ,, |
| ,, 10 | Asumani | ,, | 25 | 1st ,, | 2 | ,, |
| ,, 10 | Kainavidi | ,, | 20 | 1st ,, | 1 | ,, |
| ,, 10 | Moosura Madunga | ,, | 30 | 1st ,, | 2 | ,, |
| ,, 10 | Msabwa | ,, | 30 | 1st ,, | 1 | ,, |
| ,, 10 | Adam | ,, | 30 | 2nd ,, | 1 | ,, |
| ,, 13 | Nonbi | Female | 30 | 1st ,, | 2 | ,, |
| ,, 13 | Benjamini | Male | 24 | 1st ,, | 5 | ,, |
| ,, 13 | Kiagoffu | ,, | 30 | 1st ,, | 1 | ,, |
| ,, 13 | Kitaroma | ,, | 20 | 1st ,, | 1 | ,, |
| ,, 14 | Nateneri | ,, | 25 | 1st ,, | 1 | ,, |
| ,, 14 | Mutaisa | ,, | 15 | 1st ,, | 1 | ,, |
| ,, 14 | Erissa | ,, | 20 | 1st ,, | 1 | ,, |
| ,, 14 | Bagwibwa | Female | 18 | 1st ,, | 1 | ,, |
| ,, 14 | Johana | Male | 20 | 1st ,, | 1 | ,, |
| ,, 14 | Mwasa | Female | 18 | 1st ,, | 1 | ,, |
| ,, 14 | Rukina | Male | 25 | 2nd ,, | 1 | ,, |
| ,, 20 | Matasa | ,, | 24 | 1st ,, | 4 | ,, |
| May 4 | Kiagabidoia | ,, | 50 | 1st ,, | 5 | ,, |
| ,, 14 | Divarana | ,, | 14 | 1st ,, | 1 | ,, |

### 2. *Does the Trypanosoma occur in the Cerebro-spinal Fluid of Cases of Sleeping Sickness in other districts than Uganda?*

It will be seen from the work of the first Commission* that all or almost all cases of Sleeping Sickness examined in Entebbe, showed the presence in the blood of *Filaria perstans*, whereas cases in Kavirondo to the east of the Lake had practically no Filaria in their blood.

---

* 3, 'Distribution of Sleeping Sickness, *Filaria perstans*, etc., in East Equatorial Africa,' by Dr. Christy; and 5, 'Sleeping Sickness in its Clinical Aspects,' b Drs. Low and Castellani (both in the press).

It might possibly turn out that every case of sleeping sickness admitted to the Entebbe Hospital from the surrounding districts, harboured the trypanosoma, while cases in Kavirondo showed none.

Dr. Wiggins who is stationed at Kisumu in Kavirondo, and is in charge of a Sleeping Sickness Hospital there, kindly undertook to examine cases for the Commission. He had been in Entebbe for a fortnight at the beginning of April, and had been most energetic in examining cases with the Commission.

The following letter gives the result of his work in Kavirondo:—

The Principal Medical Officer,
East Africa and Uganda Protectorates,
Entebbe.

Kisumu,
14.5.03.

Sir,

I have the honour to forward herewith a table of my results so far as to the presence of trypanosomes in spinal fluid. Analysed they are as follows:—

| | Number examined. | Trypanosoma present. |
|---|---|---|
| Not Sleeping Sickness at all (controls) | 5 | 0 |
| Not yet diagnosed........................ | 4 | 0 |
| Sleeping Sickness, 1st stage ............ | 25 | 20 |
| ,, ,, 2nd ,, ............ | 13 | 13 |
| ,, ,, 3rd ,, ............ | 7 | 7 |

which gives—

80 per cent. of 1st stage.
100 ,, 2nd ,,
100 ,, 3rd ,,

I have a few more patients just admitted which I have not yet examined.

It is noticeable how many more trypanosomes there are present in the cases here than in Entebbe; though in a few cases there are *very* few. Yesterday, in an undoubted case, I had to examine five slides before I found a trypanosome, so that I shall examine my negative results again.

The "diagnosis" means my own diagnosis before puncture.

I have the honour to be, Sir,
Your most obedient servant,
C. A. Wiggins,
Medical Officer.

The trypanosomes are, therefore, practically present in the cerebro-spinal fluid of all cases of Sleeping Sickness in Kavirondo, as well as in Uganda, and this, which seemed to us an important question, has been satisfactorily answered.

### 3. *Does the Cerebro-spinal Fluid of the General Population contain Trypanosomes?*

Naturally it is not easy to find opportunities for performing lumbar punctures among the general population.

The following table shows the result of such controls made up to the present. More will be added as they are obtained:—

Table II.

| Date. | Name. | Sex. | Age. | Locality. | Trade. | Disease. | Trypanosoma. |
|---|---|---|---|---|---|---|---|
| 1903. | | | | | | | |
| Mar. 24 | Landu | M. | .. | Hospital | Marine | Suppurating fem. glands | Absent. |
| ,, 30 | Zake | M. | 25 | ,, | Patient | Swelling under pectoralis | ,, |
| ,, 30 | Icongo | M. | .. | ,, | ,, | .. | ,, |
| ,, 30 | Pio | M. | 12 | ,, | ,, | Fracture | ,, |
| ,, 30 | Kapere III | M. | 25 | ,, | ,, | Itch | ,, |
| ,, 30 | Eliza | F. | 18 | ,, | ,, | Yaws. | ,, |
| ,, 31 | Bofralour | M. | 16 | ,, | ,, | Pleuritis | ,, |
| ,, 31 | Zanabu | F. | 30 | ,, | ,, | Headache | ,, |
| Apr. 1 | Nabujam | F. | 45 | ,, | ,, | Cerebral tumour | ,, |
| ,, 8 | Kamsa Mahomed | M. | 25 | ,, | ,, | Madura foot | ,, |
| ,, 23 | Daudi | M. | .. | Entebbe | Prisoner | Patient in civil hospital | ,, |
| ,, 24 | Nathaniel | M. | .. | Hospital | Patient | Not diagnosed | ,, |
| ,, 29 | Arkadi | M. | .. | ,, | ,, | Suppurating bubo | ,, |
| May 8 | Matea | M. | .. | ,, | ,, | Circumcision | ,, |
| ,, 6 | Kavera | M. | .. | ,, | ,, | Rheumatism | ,, |

From this table it would appear that the cerebro-spinal fluid taken by lumbar puncture during life from members of the general population not suffering from Sleeping Sickness, does not contain the trypanosoma.

### 4. *Does the Blood of Sleeping Sickness Cases also contain Trypanosomes?*

If the cerebro-spinal fluid contains trypanosomes, they must, one would think, get into that fluid by way of the blood, and, therefore, the blood must also contain them. In the case of the blood the presence of the corpuscles is a barrier to finding the trypanosome easily. It was thought that centrifuging the blood would drive the parasites down with the red blood corpuscles. On trial this was found not to be so. It is a curious fact that both filariæ and trypanosomes resist the centrifugal action and are most readily found after being centrifuged three or even four times. The procedure adopted was to collect 10 c.c. of blood from a vein in a test-tube containing a little citrate of

Table III.

| Date. | Name. | Sex. | Age. | Duration of disease. | Trypanosoma. |
|---|---|---|---|---|---|
| 1903. | | | | | |
| Apr. 16 | Benjamini | Male | 28 | 1st stage | Present. |
| ,, 18 | Esaka | ,, | 28 | 1st ,, | ,, |
| ,, 18 | Waiswa | ,, | 10 | 1st ,, | ,, |
| ,, 18 | Kidorme | ,, | 20 | 2nd ,, | ,, |
| ,, 18 | Zebuganza | ,, | 40 | 1st ,, | ,, |
| ,, 18 | Budara | ,, | 22 | 2nd ,, | ,, |
| ,, 20 | Kimbra | ,, | 30 | 2nd ,, | ,, |
| ,, 20 | Matasa | ,, | 24 | 1st ,, | ,, |
| ,, 21 | Seera | ,, | 25 | 2nd ,, | Absent. |
| ,, 22 | Warosansa | ,, | 32 | 2nd ,, | Present. |
| ,, 22 | Katola | ,, | 25 | 1st ,, | ,, |
| ,, 27 | Koagoffa | ,, | 30 | 1st ,, | ,, |
| ,, 27 | Kitaroma | ,, | 20 | 1st ,, | ,, |
| May 12 | Nakaiba | Female | 8 | 1st ,, | ,, |
| ,, 12 | Musa | Male | 20 | 1st ,, | ,, |
| ,, 14 | Diwarana | ,, | 14 | 1st ,, | ,, |

potash solution to prevent coagulation. This was centrifuged for 10 minutes. The clear layer of plasma was now poured off and again centrifuged. This procedure was repeated four times and the sediment remaining after each centrifuging, examined microscopically.

It seemed needless to pursue this subject further, as it was evident that practically every case of Sleeping Sickness contained the trypanosome in the blood as well as in the cerebro-spinal fluid. In all probability they also could be found in the urine in some of the cases, but as little was to be gained it was not thought necessary to test this point. If the presence of the trypanosome in the blood or cerebro-spinal fluid becomes in the future a method of diagnosing this disease in doubtful cases, then it is easy to draw off 10 c.c. of either fluid.

### 5. *Does the Blood of the General Population contain Trypanosomes?*

On the West Coast of Africa, during the past two years, trypanosomes have been found in the blood of several persons. They have not been confined to the blood of natives, but at least three white people have been affected. No suspicion of these being cases of Sleeping Sickness has been entertained by those in medical charge of them. The symptoms are different. Trypanosome fever appears to cause little or no inconvenience to those harbouring the parasite, whereas in Sleeping Sickness the disease is a well-marked one and is invariably fatal. Can there be any connection between the two?

During the last few weeks Dr. Baker, of the Colonial Service, who is stationed here, has discovered trypanosomes in the blood of five natives, and Dr. Moffat, C.M.G., Principal Medical Officer of East Africa and Uganda Protectorates, has also found them in the blood of a European.

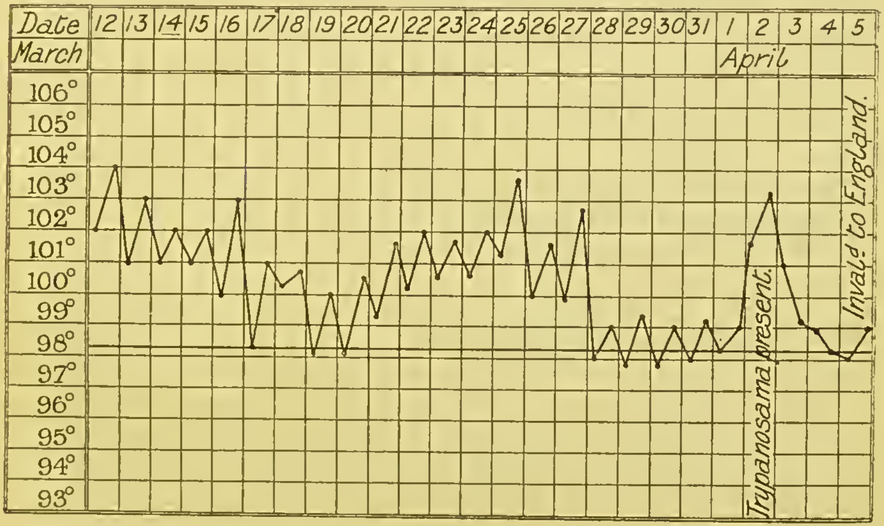
As we are face to face with a curious problem, we will, with the permission of Drs. Moffat and Baker, give a temperature chart and a short description of each of these cases. The subject, beyond its possible connection with Sleeping Sickness, is an interesting and comparatively new one.

### Case 30.—J. M. European (Male).

This case was under the care of Dr. Moffat and was treated for malaria. No malarial parasites were seen at any time, but of course the patient was under quinine. On April 2, Dr. Moffat discovered trypanosomes in the blood. It is impossible to say whether the temperature curve is due to malaria or trypanosoma. The difference between the temperature charts of the natives and the European may be a question of race.

This patient was employed in the Botanical Garden, Entebbe, which is situated on the shores of the lake and is full of tsetse flies and rank vegetation.

The following chart represents the course of the disease :—



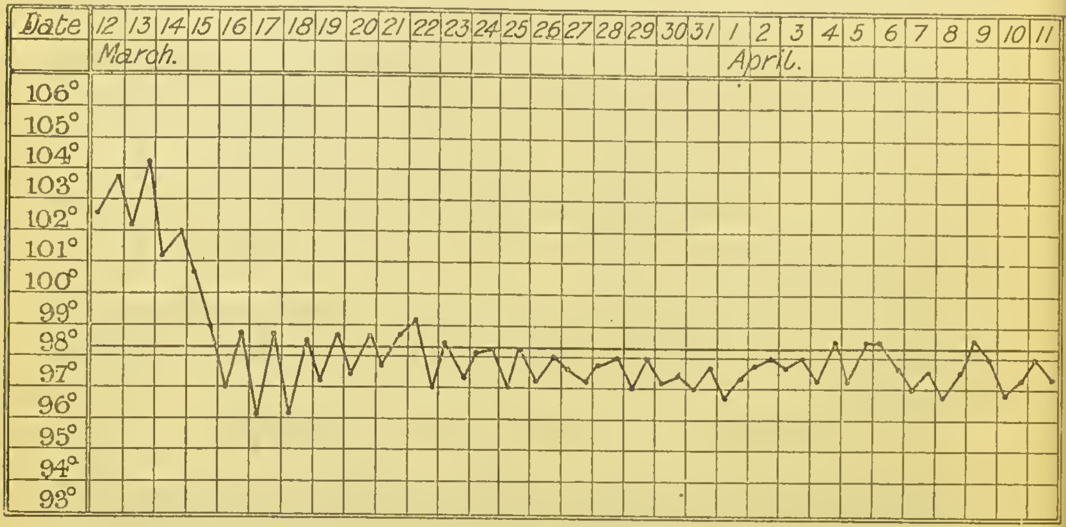
*Remarks.*—This patient has been invalided to England where the case will probably be under observation.

The trypanosomes from this case were short and plump (fig. 5, Plate 3), and quite different in appearance from those found in the blood of the natives.

### Case 31.—Karala Barigi (Male). Trypanosoma fever. District, Singo. Occupation, Policeman. Tribe Mundu, Nubian.

April 24. Patient has been quartered during the last 6 months in Entebbe. His illness began on March 10. He states that at present he feels quite well, and has no headache or other symptom. There are no enlarged glands in the neck; but in the axilla they are as large as peas, and they are also enlarged in the inguinal region. His tongue is moist and furred. His speech is fluent. Pulse 120, fair. His heart sounds are normal.

The following chart represents the course of the temperature :—



May 29. The temperature has remained normal to the present date.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 12.... | | | + | | |
| ,, 13.... | | | + | | |
| ,, 14.... | | | + | | |
| ,, 15.... | | | + | | |
| ,, 24.... | | | | | — |
| Apr. 17.... | | | | — | — |
| May 6 ... | | | | — | — |
| ,, 16.... | + | | — | | |
| ,, 19.... | | | + | | |

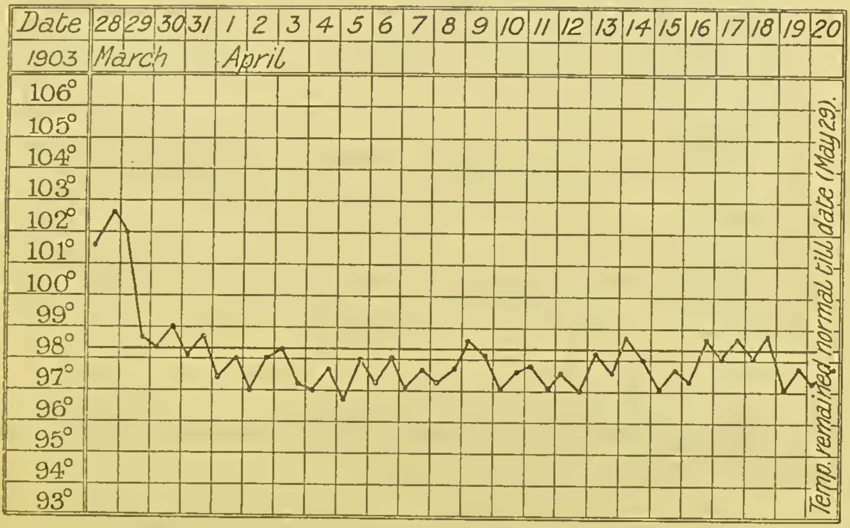
*Remarks.*—Here, then, we have a native with trypanosoma in his blood, and he shows no symptoms except the initial fever. The parasites were found in the blood in small numbers in ordinary microscopical preparations, without the previous use of the centrifuge.

It will be also noted that in spite of several lumbar puncturings the micro-organisms are not found in the cerebro-spinal fluid. The man is at duty and seems to be in perfect health.

Case 63.—Kumsarsàbba (Male). Age 25 years. Trypanosoma fever.
District, Buddu. Occupation, Policeman.

March 28, 1903. Was admitted to hospital. He states that he has been in the force 11 months, that none of his people have had sleeping sickness, and that he has never been ill like this before. He has no œdema, walks well, the lymphatic glands in the neck are not enlarged; there is some slight enlargement in the glands of the groin. His tongue is furred. Pulse 120. Heart sounds normal. He has some tremor of hands and tongue. His speech is normal.

The following chart reports the course of the disease:—



May 29. The temperature remained normal to present date.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

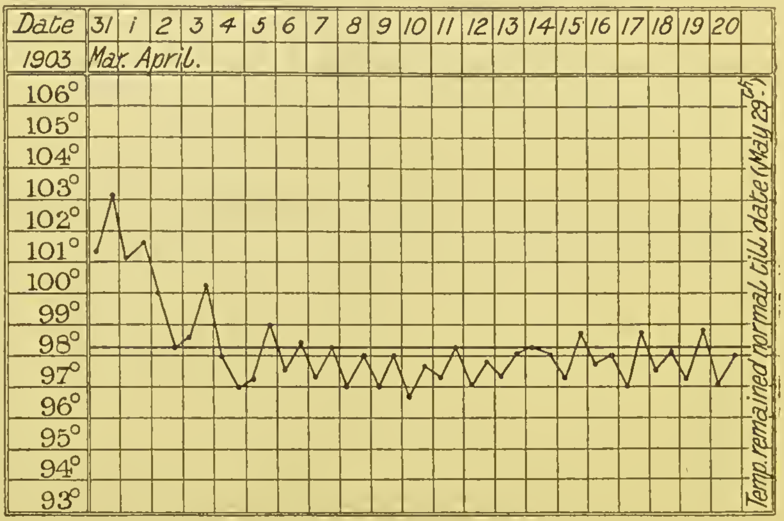
| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 26.... | | | + | | |
| Apr. 17.... | | | | – | – |
| May 7.... | | | | – | |
| „ 16.... | | | – | | |
| „ 19 ... | + | | – | | |

*Remarks.*—This man is also at duty.

**Case 64.**—Jordien Murjan (Male). Age 35 years. Trypanosoma fever. District, Muru, Nubian. Occupation, prisoner, for last 2 years.

March 31, 1903. Admitted to hospital. He is an old Soudanese mutineer and lives in the jail. No fellow prisoners have had Sleeping Sickness. He has no œdema, and no noticeable swelling of glands. His tongue is healthy, but shaky. There is no tremor of the hands. His speech is normal and pulse 144.

The following chart represents the course of the disease:—



The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 31.... | | | + | | − |
| Apr. 1.... | | | + | | |
| „ 2.... | | | + | | |
| „ 3.... | | | + | | |
| „ 17.... | | | | − | − |
| May 1.... | − | − | + | | − |
| „ 11.... | | | + | | − |
| „ 25.... | | | + | | − |

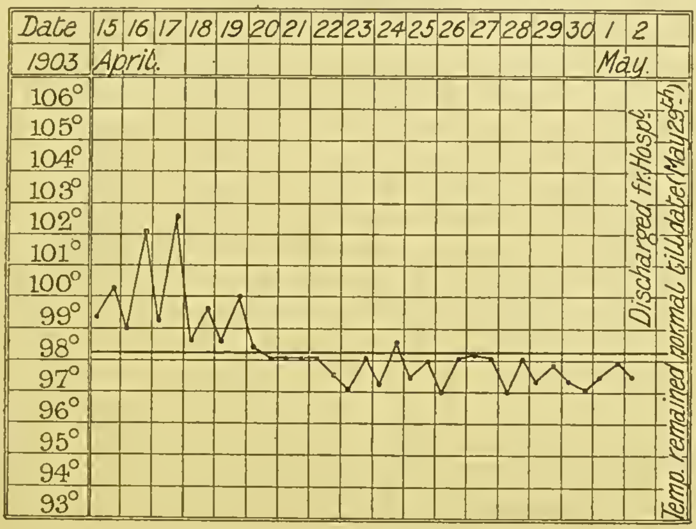
This man has been repeatedly examined by lumbar puncture with negative results.

CASE 66.—Tabula (Male). Age 25. Trypanosoma fever. District, Marine Village, Entebbe. Occupation, Marine.

April 15, 1903. Admitted to hospital. He states that he has been in the service one year. He has never had this disease before. There are no sleeping sickness cases in the marine village.

April 24. Patient complains of a slight headache to-day. His pulse is 96, his heart is normal. The glands at the back of his neck are slightly enlarged, as are also those of the inguinal region. His speech is normal, he sleeps well, and his tongue is moist and clean. He has no tremors.

The following chart represents the course of the disease:—



The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

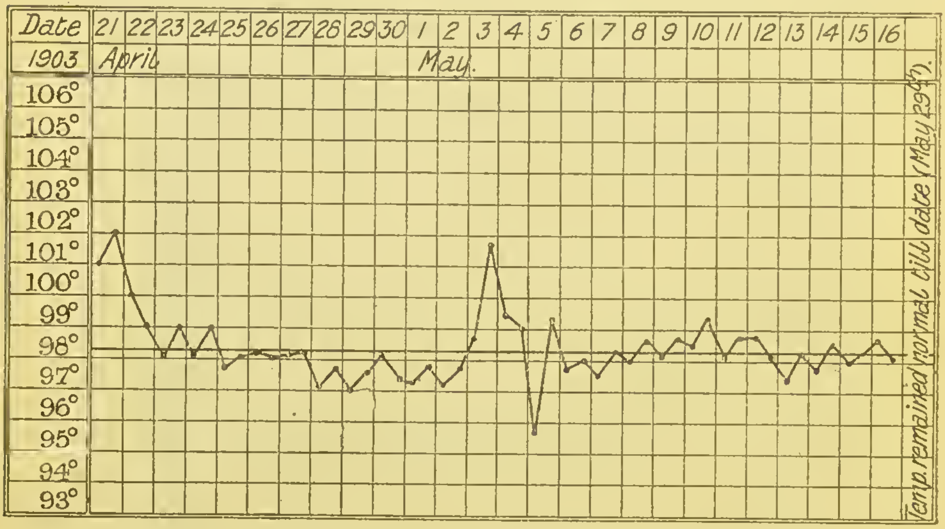
| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 15.... | – | – | + | | – |
| „ 17.... | | | | + | – |
| May 6.... | | | | – | – |
| „ 20.... | + | | + | – | – |

CASE 68.—Bara Risgallah (Male). Age 35 years. Trypanosoma fever. Tribe Lendu. Occupation, Police. Lives in hut in police lines.

April 21, 1903. Admitted to hospital. This man states he has been ill 10 days, and that the ailment began with a shivering fit. He also says he had a sickness like this in Kampala 4 or 5 years ago.

April 24. Patient looks ill. There is no œdema, and only the glands in the groin are slightly enlarged His pulse is 108, feeble, compressible. The heart sounds are normal. He has no tremors.

The following chart represents the course of the disease:—



The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 12.... | + | | + | | – |
| May 4.... | | | + | | – |
| „ 11.... | | | + | | |

From these cases it will be seen that these trypanosomes do not visibly affect the health of their native hosts. All the men are at duty and say they feel strong and well. It is a curious fact that although the parasites are found in the blood they have never been seen in the cerebro-spinal fluid. The question seems to resolve itself into this: either the trypanosomes found in sleeping sickness and those found in

Table IV.—List of Controls up to Present Time.

| Date. | Name. | Trade. | Sex. | Centrifuged. | Filaria. | Trypanosoma. | |
|---|---|---|---|---|---|---|---|
| 1903. | | | | | | | |
| Apr. 23 | Ainara | Prisoner | Male | 10 c.c. | + | – | Healthy. |
| " 23 | Biggity | " | " | " | + | – | " |
| " 24 | Nathaniel | Dispensary | " | " | – | – | Case in hospital. |
| " 28 | Chia Msoga | " | " | " | + | – | " " |
| " 29 | Aradiki | " | " | " | + | – | " " |
| " 29 | Arkadi | " | " | " | + | – | " " |
| " 29 | Mandandiki | " | " | " | – | – | " " |
| May 6 | Jumabini | Prisoner | " | " | – | – | Healthy. |
| " 8 | Matea | Dispensary | " | " | + | – | Circumcision. |
| " 13 | Bifarawala | Prisoner | " | " | + | – | Healthy. |
| " 16 | Amuri Abdulla | " | " | " | – | – | " |
| " 18 | Serewame | " | " | " | – | – | " |
| " 21 | Juma Bin Abdullah | " | " | " | – | – | " |
| " 22 | Baraca Bin Salimi | " | " | " | – | – | " |
| " 27 | Musa | " | " | " | + | – | " |
| " 27 | Weraga | " | " | " | + | – | " |
| " 28 | Wanika | " | " | " | + | – | " |
| " 29 | Baki | " | " | " | + | – | " |

trypanosoma fever belong to different species and give rise to different diseases, or they are one and the same, and if confined to the blood give rise to slight feverish symptoms, whereas if they gain entrance to the cerebro-spinal fluid they give rise to sleeping sickness. The question cannot be answered at present, but doubtless a solution will be found before long.

But with the exception of these six cases who came for medical treatment on account of fever, no trypanosomes have been found in the blood of the general population. (See Table on p. 23.)

### 6. *Can any Difference be made out Microscopically between the Trypanosoma of Sleeping Sickness and that of Trypanosoma Fever?*

The trypanosomes have been so often described of late years, and are beginning to be so well known, that it is unnecessary for us to give a general description of them.

In order to make out if any difference in shape and size exists between the trypanosoma of sleeping sickness and of trypanosoma fever, ten parasites were measured in each of ten cases and an average taken. The length, the distance of the micro-nucleus from the posterior extremity and the presence or absence of chromatic dots were the points fixed on.

The following table shows the results:—

Table V.—Trypanosomes of Sleeping Sickness.

| Name. | Length. | Chromatic dots. | Distance of micro-nucleus from posterior end. |
|---|---|---|---|
| | μ | Per cent. | μ |
| Zakubu............ | 21·5 | 10 | 0·5 |
| Waiswa........... | 19·0 | 70 | 0·2 |
| Kidorme .......... | 20·4 | 10 | 0·4 |
| Katola............. | 26·1 | 10 | 1·0 |
| Warasansa ......... | 21·8 | 0 | 0·25 |
| Average..... | 21·7 | 20 | 0·47 |

Table V.—*continued.*—Trypanosoma Fever.

| Name. | Length. | Chromatic dots. | Distance of micro-nucleus from posterior end. |
|---|---|---|---|
| | μ | Per cent. | μ |
| J. M. .............. | 17·8 | 0 | 1·0 |
| Jordien Murjan..... | 28·0 | 0 | 2·1 |
| Bara Risgallah...... | 27·4 | 20 | 1·4 |
| Karala Barigi....... | 25·2 | 0 | 1·1 |
| Tabula ............ | 23·2 | 0 | 2·1 |
| Average..... | 24·3 | 4 | 1·5 |

From Table V it would appear that the trypanosomes found in sleeping sickness are shorter, have chromatic dots more frequently, and the micro-nucleus is situated nearer the end than in the other. Whether this should have any weight in determining the question as to specific difference it is difficult to say. At present it seems better to be content with noting facts and wait for further knowledge. In the *Trypanosoma Brucei* differences in size and shape were noted in different animals, and Bradford and Plimmer state that differences are seen at different stages of the disease in the same animal. It is curious that the shortest form is found in J. M. who was supposed to be suffering from trypanosoma fever, and a monkey inoculated from him showed the same short fat forms.

We attach a few rough sketches of trypanosomes from various cases (Plates 1—3).

### 7. *Can any Difference be made out between these Trypanosoma by Experiments?*

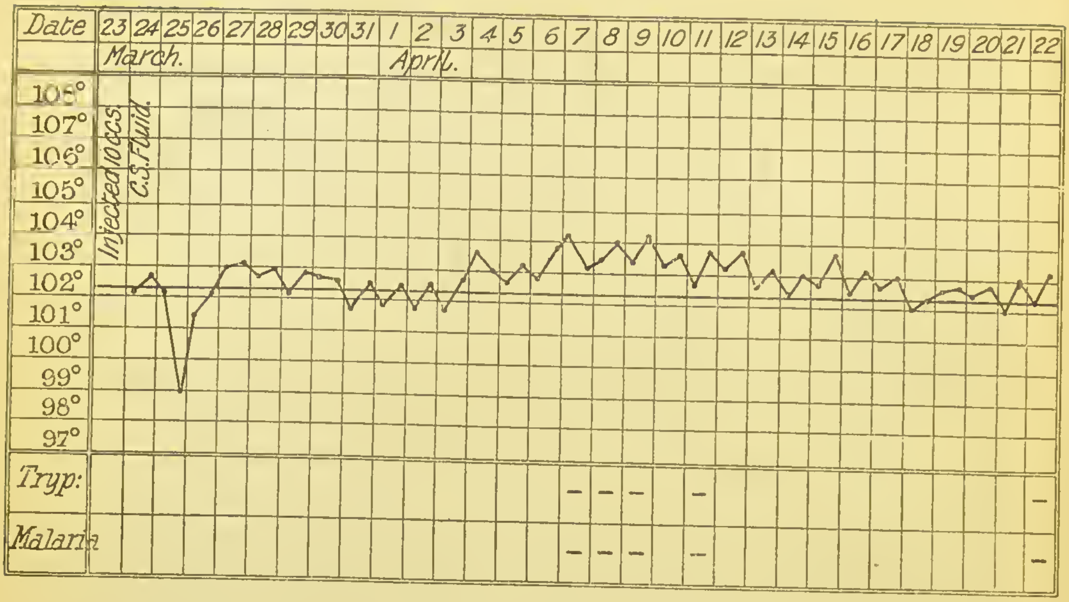
### The Trypanosoma of Sleeping Sickness in the Monkey.

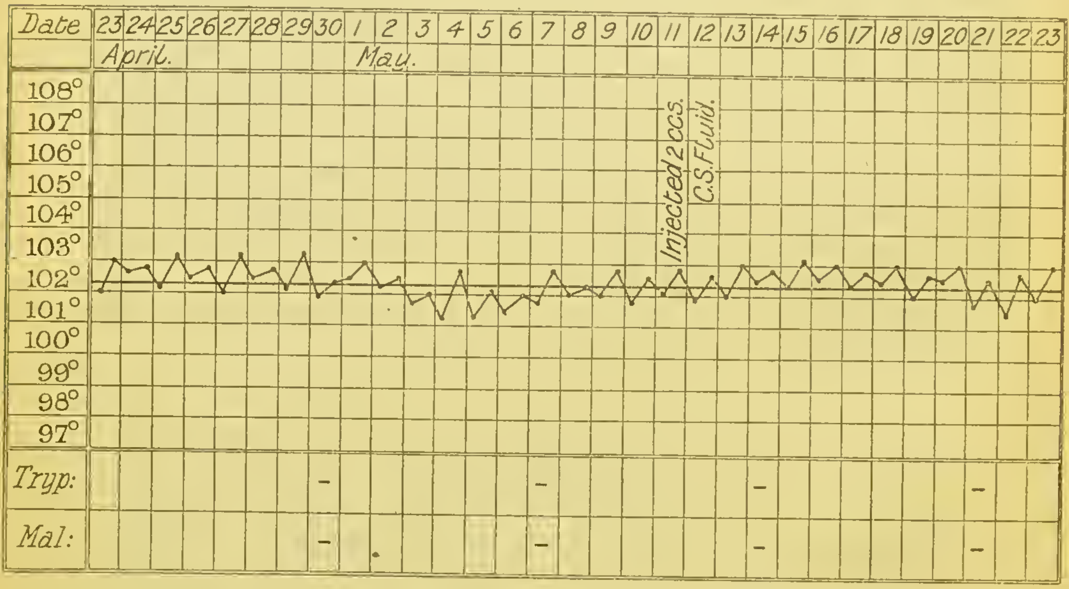
Experiment 1.—Monkey (Male). Pale-faced variety.

To note the effect of the subcutaneous injection of cerebro-spinal fluid containing trypanosomes.

March 23, 1903. Injected 10 c.c. of cerebro-spinal fluid taken, *post mortem*, from Case 18, Kaperi, under the skin of the left side of monkey.

The following chart represents the temperature and the presence or absence of malaria and trypanosoma:—





May 28. Malaria absent. Trypanosomes *present.*

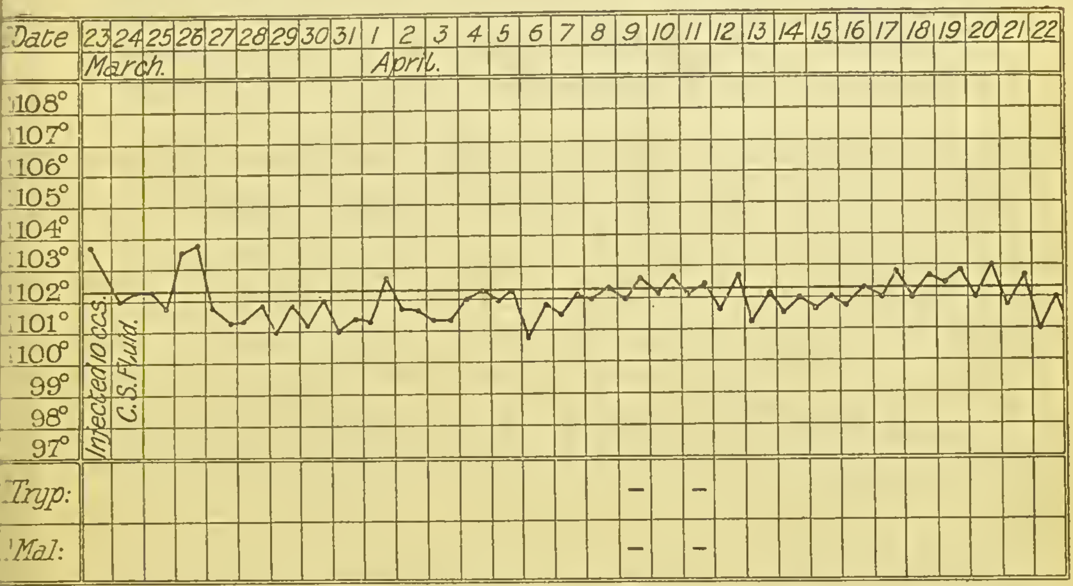
*Remarks.*—The pale-faced variety of monkeys were brought by us from England. We do not know the name of the species, but the attached photograph (Plate 4, fig. 7) will show. None of these English monkeys harbour malaria parasites, as most of the local species do.

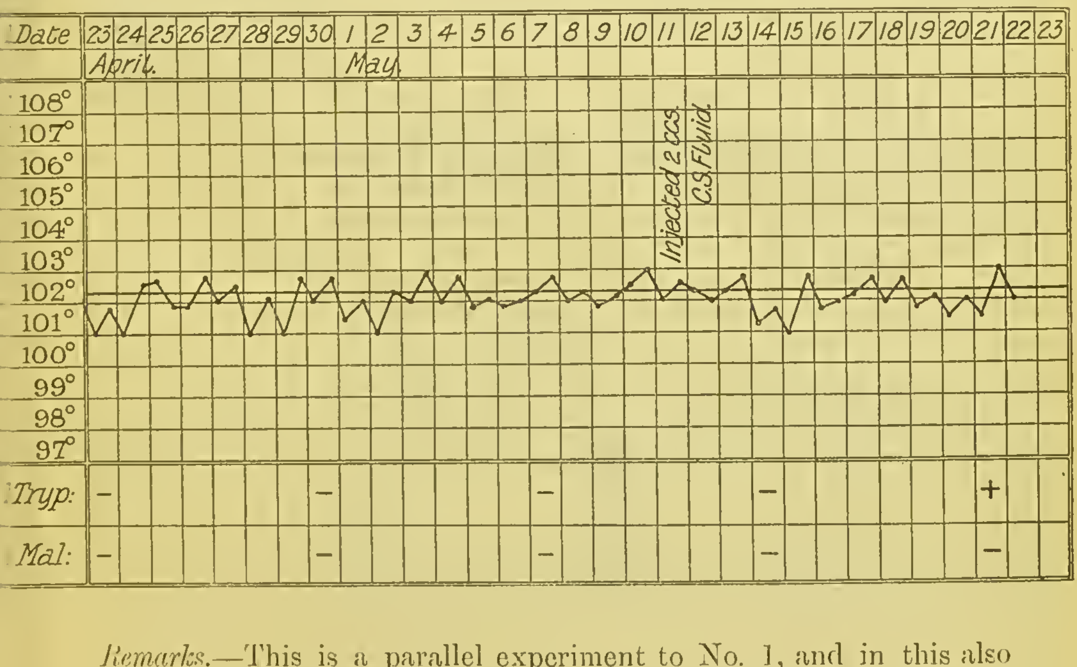
The first injection of cerebro-spinal fluid evidently did not contain any living trypanosoma. This may be due to the fact that the fluid was taken *post mortem,* and the trypanosomes soon disappear in the dead body. On May 11, the monkey was re-injected and the parasite appears in the blood 17 days later. The temperature chart is given to show the normal temperature of a monkey.

EXPERIMENT 2.—Monkey (Male). Pale-faced variety.

March 23, 1903. Injected sediment of about 10 c.c. of cerebro-spinal fluid, taken *post mortem*, from Case 18, Kaperi.

The following chart represents the temperature and the presence or absence of malaria and trypanosoma:—





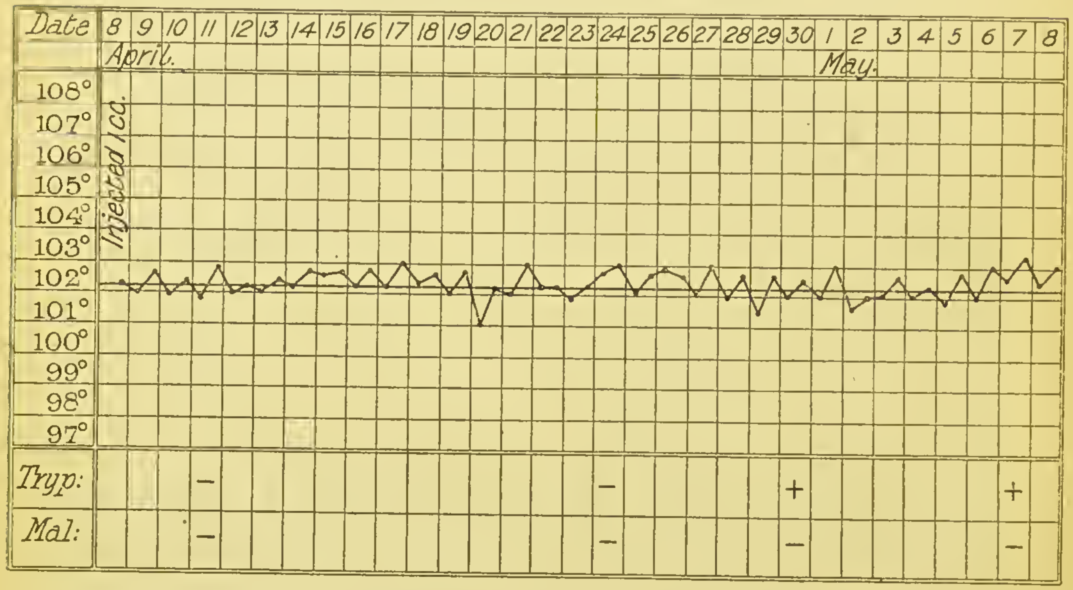
*Remarks.*—This is a parallel experiment to No. 1, and in this also the first injection remained barren. Ten days after the second injection, the trypanosomes appear in the blood. This appearance of the trypanosomes is not heralded by any change in the temperature, and the monkey shows no signs of disease.

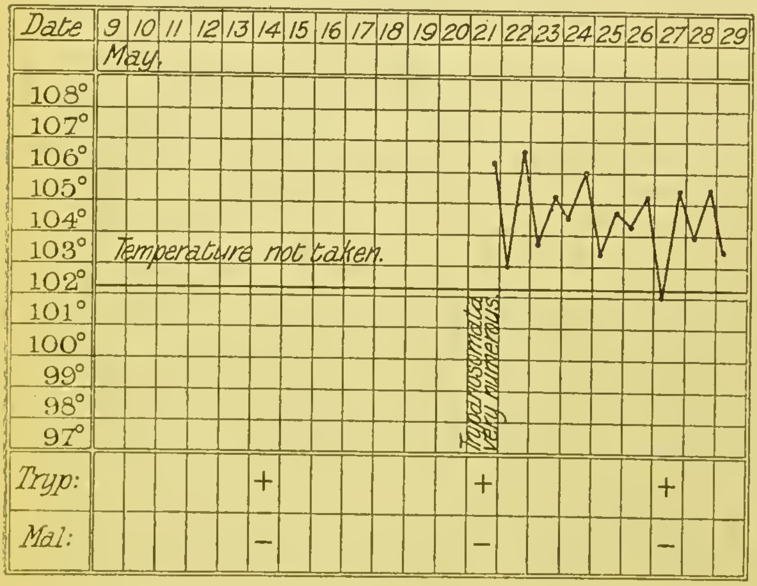
### Experiment 34.—Monkey (Male). Pale-faced variety.

To note the effect of injecting the cerebro-spinal fluid from a case of sleeping sickness into the vertebral canal of a monkey.

April 8, 1903. Injected 1 c.c. of cerebro-spinal fluid containing trypanosomes from a case of sleeping sickness into the spinal canal of this monkey.

The following chart represents the temperature and the presence or absence of malaria and trypanosoma:—





*Remarks.*—The chief point of interest in this case is the increase in number of the trypanosomes about May 21, and the rise of temperature at the same time. It cannot be said that this animal shows any symptoms of sleeping sickness up to the present time.

EXPERIMENT 95.—Monkey. Black-faced variety. (Plate 4, fig. 8.)

May 13, 1903. Blood examined, there was no malaria or trypanosoma.

May 14. Injected 1 c.c. of cerebro-spinal fluid from a case of sleeping sickness into the vertebral canal of this monkey.

May 22. Trypanosoma absent. Malaria absent.

„ 29. „ „ „ „

EXPERIMENT 96.—Monkey. Black-faced variety.

May 14, 1903. Blood examined. No malaria, no trypanosoma.

„ Injected 1 c.c. of cerebro-spinal fluid from a case of sleeping sickness into the vertebral canal of this monkey.

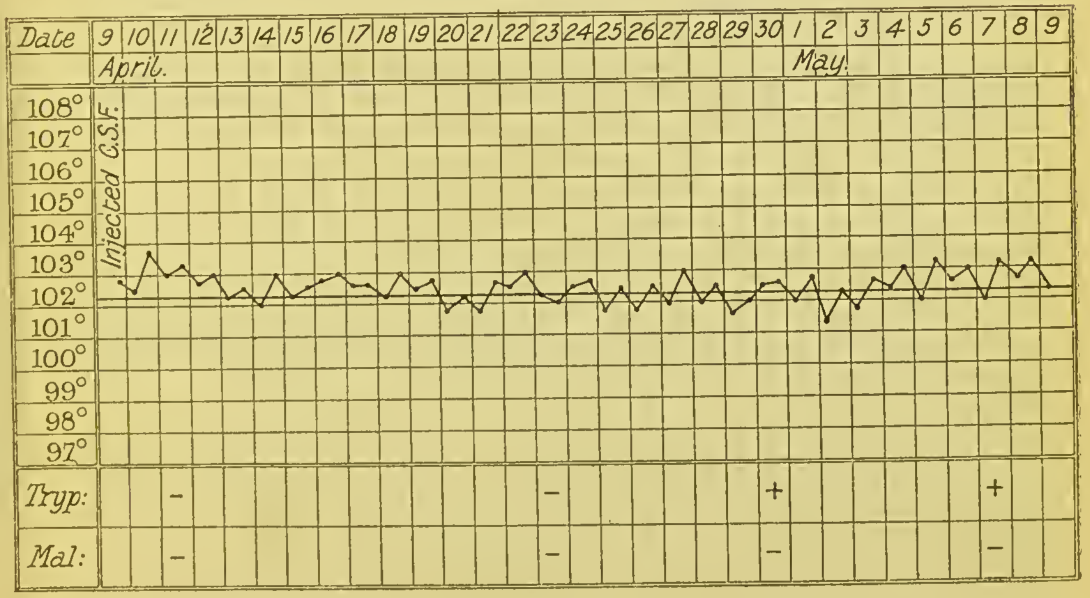
May 22. Trypanosoma absent. Malaria present.

„ 29. „ „ „ „

*Remarks.*—After 15 days the trypanosomes are still absent from the blood.

EXPERIMENT 54.—Monkey. Black-faced variety.

To note the effect of injection of cerebro-spinal fluid containing trypanosomes, from a case of sleeping sickness, into the brain cavity of a monkey.



April 9, 1903. Injected 1 c.c. of cerebro-spinal fluid from a case of sleeping sickness into the brain cavity of this monkey through the foramen magnum.

The preceding chart represents the temperature and presence or absence of trypanosoma and malaria.

May 14. Trypanosoma present. Malaria absent.
,, 21. ,, ,, ,, ,,
,, 28. ,, ,, ,, ,,

*Remarks.*—The trypanosomes appeared in the blood 21 days after injection.

### Experiment 98.—Monkey. Black-faced variety.

May 14, 1903. Malaria present, trypanosoma absent. Injected 1 c.c. of cerebro-spinal fluid from a case of sleeping sickness into the brain of this monkey through the foramen magnum.

May 22. Trypanosoma absent. Malaria numerous.
,, 29. ,, present. ,, present.

*Remarks.*—The parasites appeared in the blood after 15 days.

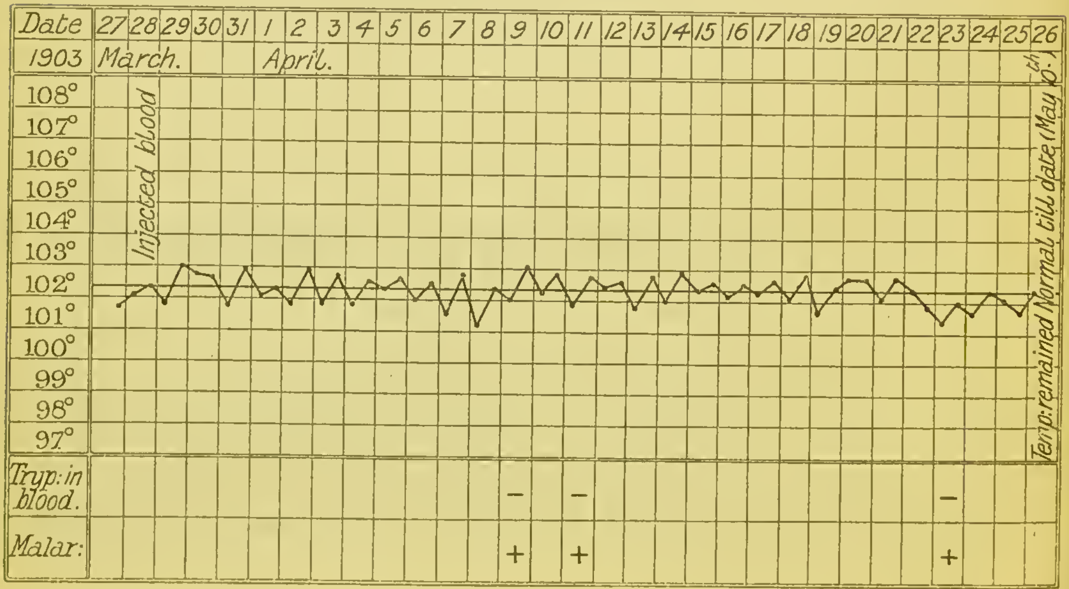
## *The Trypanosome of Trypanosoma Fever in the Monkey.*

### Experiment 6.—Monkey. Black-faced variety.

To ascertain if the trypanosome of trypanosoma fever is pathogenic to monkeys.

March 28, 1903. Injected 4 c.c. of blood from Dr. Baker's case Kumsasabba, a policeman whose blood contained trypanosomes yesterday.

The following chart represents the temperature and the presence or absence of trypanosoma and malaria:—



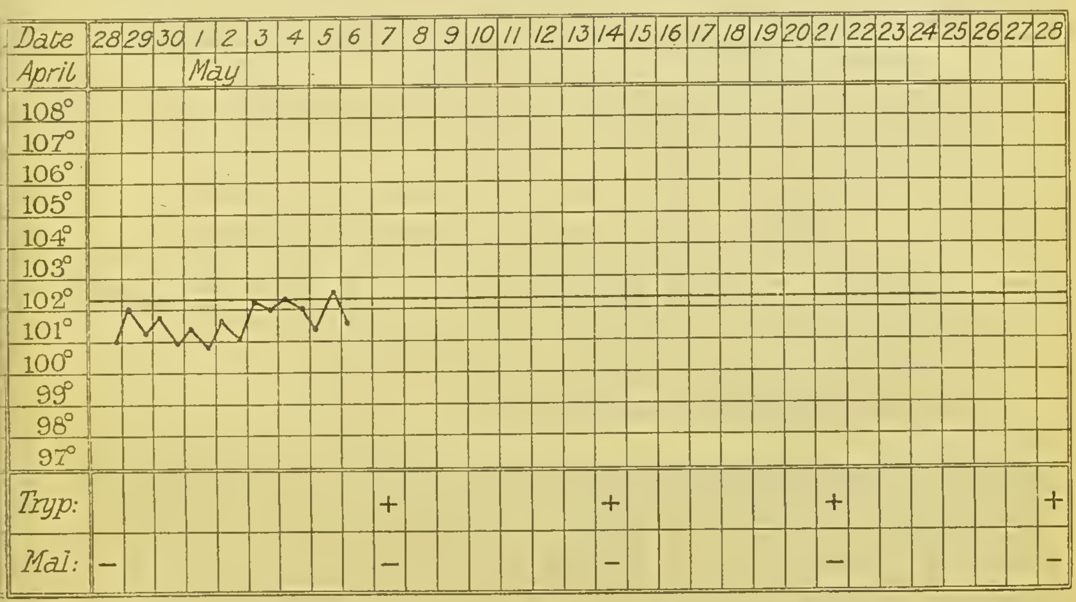
April 30. Malaria present. Trypanosoma absent.
May 7. ,, ,, ,, present.
,, 24. ,, ,, ,, ,,
,, 21. ,, ,, ,, ,,
,, 28. ,, ,, ,, ,,

*Remarks.*—The trypanosomes appeared in the blood on May 7, 40 days after the injection of blood.

### EXPERIMENT 61.—Monkey. Pale-faced variety.

April 28. Injected 2 c.c. of blood from Case 66, Tabula, marine, containing trypanosoma, into the spinal canal.

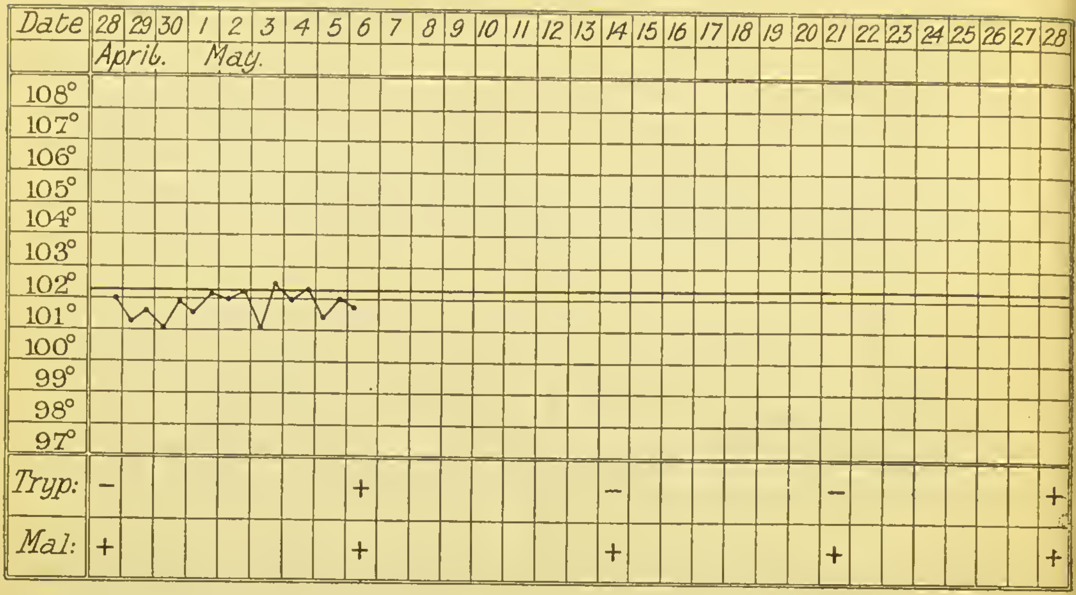
The following chart represents the temperature and presence or absence of trypanosoma and malaria :—



### EXPERIMENT 56.—Monkey. Pale-faced variety.

April 28. Injected 2 c.c. of blood from Case 66, Tabula, marine, containing trypanosoma into the spinal canal of this monkey.

The following chart represents the temperature and resence or absence of trypanosoma and malaria :—

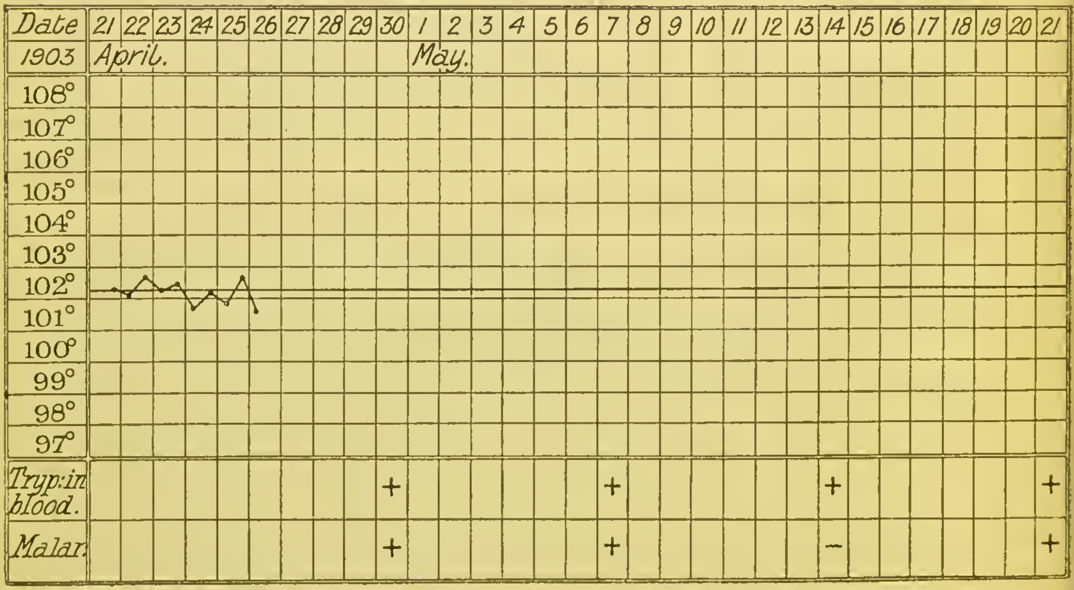


*Remarks.*—The temperatures were not continued after the first week on account of want of labour.

EXPERIMENT 58.—Monkey. Black-faced variety.

April 21. Injected subcutaneously 3 c.c. of blood, containing trypanosomes from Case 68, Bara Risgallah, marine.

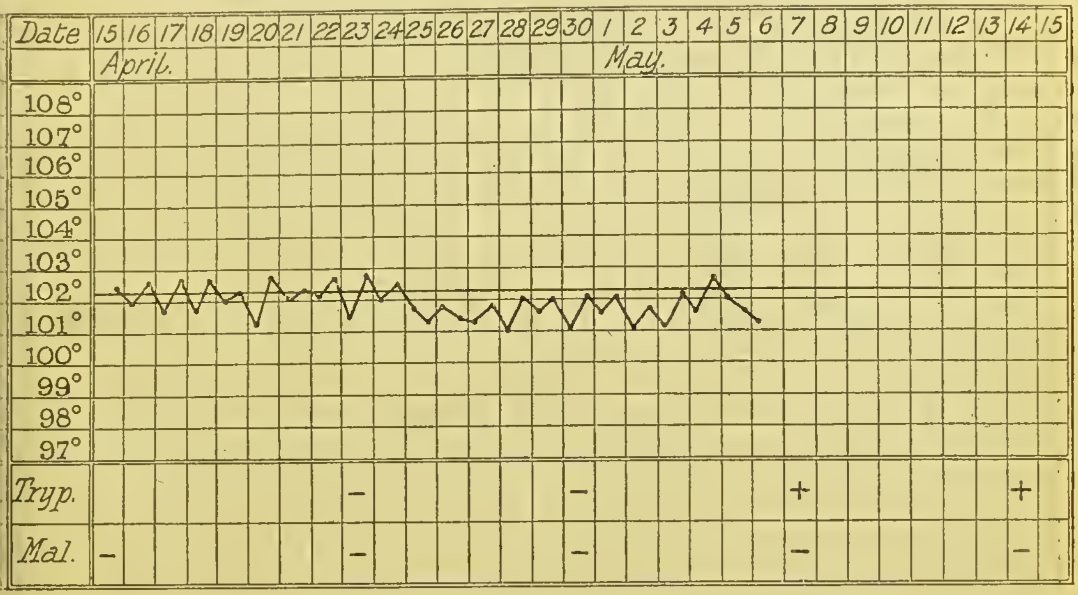
The following chart represents the temperature and presence or absence of trypanosoma and malaria:—



EXPERIMENT 60.—Monkey. Pale-faced variety.

April 15. Injected subcutaneously 2 c.c. blood from Case 66, Tabula, marine, containing trypanosoma.

The following chart represents the temperature and presence or absence of trypanosoma and malaria :—



May 21. Trypanosomes very numerous. Malaria absent.
„ 28. „ „ „ „ „

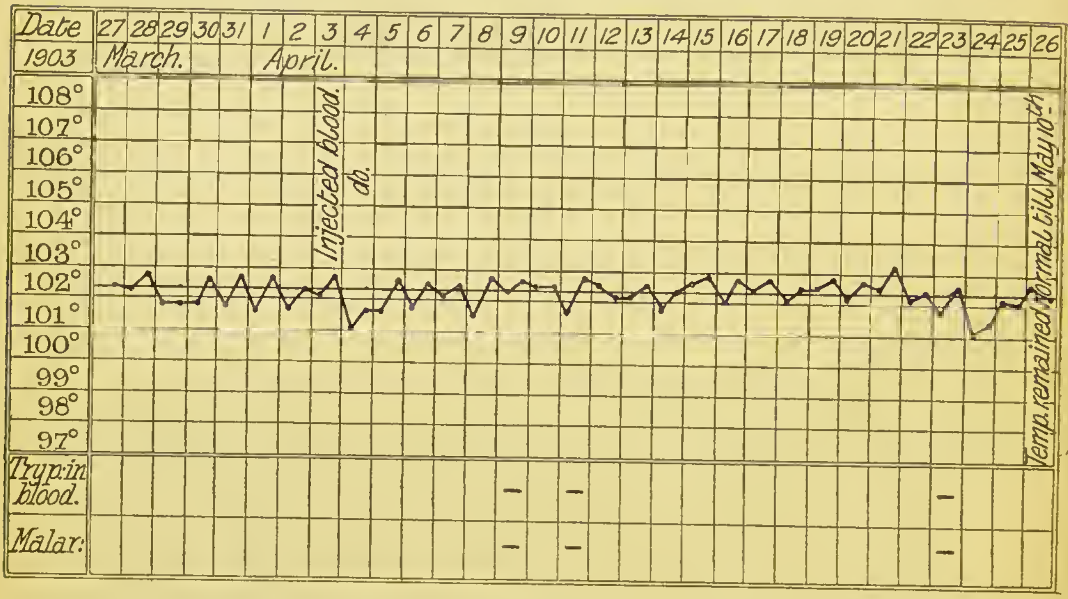
### Experiment 8.—Monkey. Black-faced variety.

April 3. Injected subcutaneously a small quantity of blood from Case J. M., whose blood is seen to contain trypanosomes this morning. The blood taken only amounted to a drop or so and had firmly clotted.

April 4. Injected about 1 c.c. of blood from same case.

May 11. Injected 2 c.c. of blood from Jordien Murjan.

The following chart represents the temperature and presence or absence of trypanosoma and malaria :—



| | | |
|---|---|---|
| April 30. | Malaria absent. | Trypanosoma absent. |
| May 7. | ,, ,, | ,, ,, |
| ,, 14. | ,, ,, | ,, ,, |
| ,, 21. | ,, ,, | ,, ,, |
| ,, 28. | ,, ,, | ,, ,, |

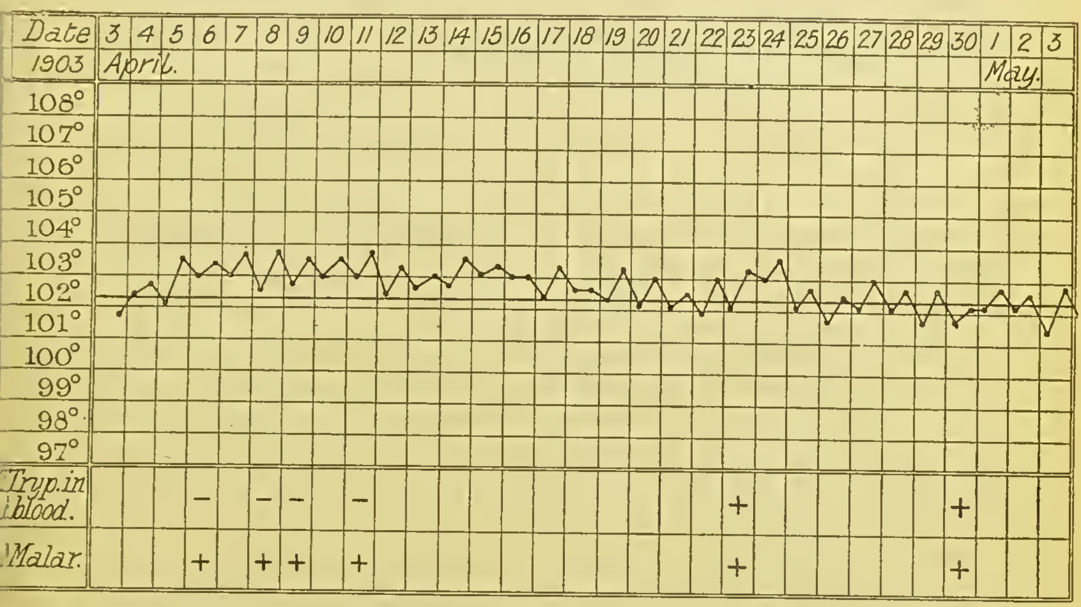
*Remarks.*—The parasites appear in the blood after 10 days, after the last injection.

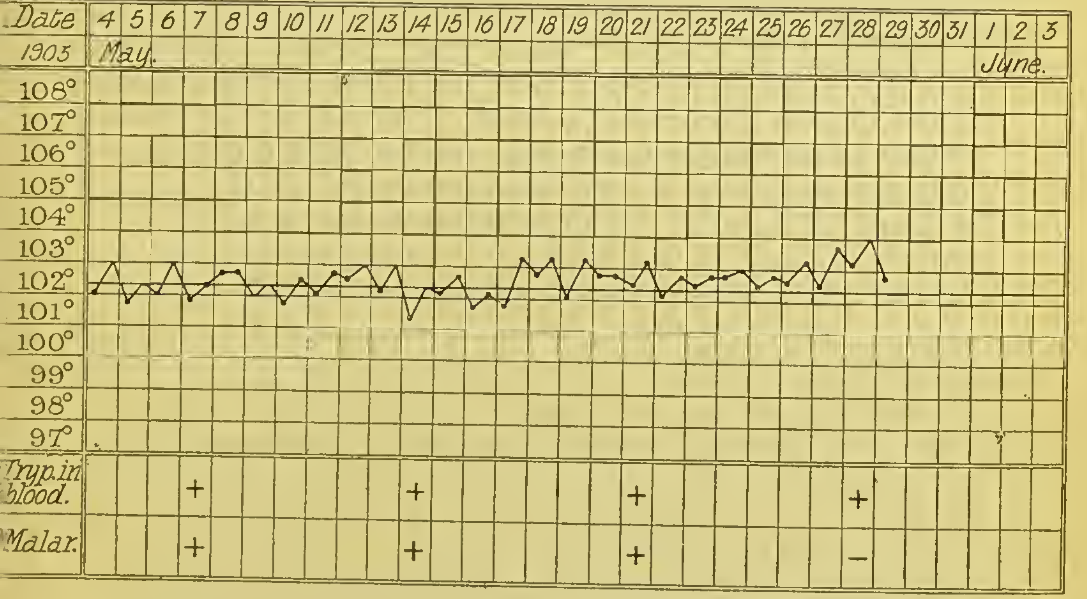
EXPERIMENT 32.—Monkey. Black-faced variety.

April 3. Injected $\frac{1}{2}$ c.c. of blood from Case 44, J. M., containing trypanosomes.

April 4. Injected 1 c.c. of blood from the same case.

The following chart represents the temperature and presence or absence of trypanosoma and malaria:—





From these experiments on the monkey, it is evident that no deduction can be made at present as to the identity or otherwise of the trypanosomes, found in cases of sleeping sickness and those found in trypanosoma fever.

Rats, guinea-pigs, dogs, sheep, goats, donkeys, and oxen are also under observation, but the results are not sufficiently definite to merit particular description.

In the rat both trypanosomes appeared on the 11th day.

In the dogs in one case the trypanosomes from a case of sleeping sickness appeared in the blood in 38 days. In two dogs inoculated with blood from a case of trypanosoma fever, the trypanosomes appeared in 13 and 19 days respectively.

## 8. *Does the Distribution of Sleeping Sickness coincide with the Distribution of any biting Insect?*

The distribution of Sleeping Sickness in Uganda is striking. Speaking broadly, the disease only occurs along the northern shores of the Victoria Nyanza, in a strip of country about 15 miles wide.

The Prime Minister of Uganda informs me that any cases which are found further inland are imported. The numerous islands which lie near the northern shores of the lake are also the seat of the disease. Between these islands and the mainland and for several miles inland, a constant interchange of commodities, and intercourse between the natives takes place.

There must be some reason for this peculiar distribution. It cannot be due to a food poison, as the natives outside this narrow strip have the same habits. Then again, if the trypanosomes are the cause of the disease, which there is every reason to suspect, they cannot be conveyed by food or clothes, or directly from man to man, but must be carried by some blood-sucking insect. Naturally on the analogy of Nagana a Tsetse Fly was suspected. But the tsetse is not supposed to be present at altitudes of 3,000 feet and upwards, nor is it supposed to be found in Uganda. A short search in the Botanical Gardens, Entebbe, disposed of these objections. A tsetse fly very similar to the one met with in the Fly Country, Zululand, was found in numbers. At present and for some time past we have hundreds of live tsetse flies brought into the laboratory every day by a couple of native children. The flies must be swarming in the vicinity of Entebbe. This gave a lead. The Regents were asked to meet the Commission and have the matter explained to them. They at once recognised the fly as one known to the Muganda as Kivu, and said that it was found along the shores of the lake.

The chiefs were supplied with several dozen nets, killing bottles and boxes, and on their part promised to have the distribution of the fly worked out. A letter was also addressed to Bishops Tucker and Hanlon, who kindly promised to obtain information on this point from the Missionaries. To each Missionary was sent a specimen of the tsetse fly and instructions as to collecting them. It is too early to give results, but up to the present the distribution of the Tsetse Fly and Sleeping Sickness agree. In a few weeks we expect to have sufficient data to enable us to make a definite statement.

* What species of tsetse fly is found here, has not been made out. There seem to be two varieties, one darker than the other, and both darker than the Zululand species. The Uganda species is also smaller than the Zululand, the males averaging 9 mm. in length and the females 10 mm., whereas the latter measured 11 mm. and 12·5 mm. respectively. It is unnecessary for us to go more fully into this question of species, since specimens have been sent to Mr. Austen of the British Museum, and by this time the Committee will doubtless have his report.

Another point in this connection must be touched upon. Dr. Moffat in travelling from Mombasa to Port Florence caught three different kinds of tsetse fly in the railway carriage, whilst passing through the coast fly belt. This raises the question as to whether the coast tsetse flies can carry the sleeping sickness trypanosoma. If they do there is danger of the disease passing into the coast fly belt. This raises the next question.

### 9. *Can the Uganda Tsetse Fly carry the Trypanosoma found in Sleeping Sickness Cases from Animal to Animal?*

This has not been proved yet, but experiments are being made which should decide the question in a short time. Curiously at this very moment word is brought that one of these experiments has proved successful, and that a most important one. We shall give it in full.

EXPERIMENT 94.—Monkey. Black-faced variety.

To ascertain if tsetse flies freshly caught in the vicinity of Entebbe are carrying trypanosomes.

May 13. Blood examined. No trypanosomes. No malaria.
,, 13. Fed 31 flies freshly caught near Entebbe.
,, 15. ,, 15 ,, ,, ,,
,, 18. ,, 10 ,, ,, ,,
,, 19. ,, 7 ,, ,, ,,
,, 19. ,, 13 ,, ,, ,,
,, 20. ,, 13 ,, ,, ,,
,, 21. ,, 16 ,, ,, ,,

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* 29.5.03.—A letter has just been received from Mr. Austen informing us that the Uganda species is *Glossina palpalis*. He states that this is a West African species, and its occurrence in Uganda is *very* interesting and was quite unexpected by him.

Blood examined. No trypanosomes. No malaria.
May 22. Fed 20 flies freshly caught near Entebbe.
,, 23. ,, 14 ,, ,, ,,
,, 23. ,, 11 ,, ,, ,,
,, 24. ,, 17 ,, ,, ,,
,, 25. ,, 14 ,, ,, ,,
,, 25. ,, 8 ,, ,, ,,
,, 25. ,, 9 ,, ,, ,,
,, 26. ,, 7 ,, ,, ,,
,, 26. ,, 11 ,, ,, ,,
,, 27. Trypanosomes *present.*

This important experiment has succeeded quicker than we anticipated. It is impossible with our present knowledge to tell if the trypanosomes, carried by the tsetse flies to the monkey, are the same as those found in cases of Sleeping Sickness, but the result is very striking.

The flies were caught in the vicinity of the hut-tax labourers' camp. These men, to the number of thousands, come to Entebbe and work for Government for 1 month in lieu of paying hut tax. They live in rudely built grass huts near the shores of the lake about a mile from Entebbe along the Kampala road. Tsetse flies caught at this place and brought in cages to the laboratory have conveyed trypanosomes to a monkey. What could be more suggestive? The Prime Minister of Uganda told us he considered the hut-tax system as the chief cause of the sowing of Sleeping Sickness all over the land.

More work is required before an opinion can be expressed.

The Commission leave the question for the present at this stage with the conviction that they have at least given the Committee some material for consultation and suggestion.

The Commission think it well even at this early stage to express their heartiest thanks to Colonel Sadler, C.B., H.M. Commissioner, Uganda, for his invariable kindness and good offices; to Dr. Moffat, C.M.G., who has been untiring in help; to Dr. Hodges, Acting P.M.O. during Dr. Moffat's absence; to Dr. Baker, the resident colonial surgeon in Entebbe, who has spent most of his spare time in working with and for the Commission; to Dr. Wiggins, of Kisumu, who has given valuable assistance; to Mr. Stanley C. Tomkins, Assistant Commissioner, who has been assiduous in sending us collections of biting flies; and lastly to the Prime Minister and Regents of Uganda, who are now making out the distribution of tsetse flies in Uganda and have throughout shown great interest in the work.

## APPENDIX.

As we understand the members of the previous Commission sent out by the Royal Society are preparing an elaborate report on the clinical features of this disease, we shall confine ourselves for the present to merely sending a few completed histories of cases which have been under our care. In our opinion the reading of a few cases will give as good an idea of the disease as is required for our purpose at the present time. It is customary to divide the disease into three stages. In the first stage there is usually fever and in rare cases symptoms of exaltation or even mania, but as a rule the patient is quiet and the facial expression is dull, otherwise there is nothing noteworthy. In the second stage, there is paresis of gait and speech, tremors of the tongue, lips and hands, and the dullness of expression deepens. In the third stage these symptoms have become more pronounced and the patient is confined to bed in a condition of complete lethargy.*

### CASE 4.—Kaperi (Male). Age 8 to 10 years.

July 15, 1902. Admitted to hospital under Dr. Castellani's and Dr. Low's care.

March 22, 1903. The temperature chart shows a rise to 100—101° F. at night during July. Later it was more irregular. In December, 1902, it rose for a week, also in January and February, 1903. It has been sub-normal during the whole of March.

At the present time this boy is very thin. There is a commencing bed sore on the right hip. He understands questions when spoken to. He lies with the thighs drawn up on the abdomen. The fingers of the right hand are flexed in the palm, and the right wrist is also kept flexed, and he is unable to extend either his fingers or wrist. He can neither stand nor walk. He complains of pain in legs and thighs. His tongue is furred and is not tremulous. No tremor of left hand. Knee-jerk is absent. He takes milk, which he swallows easily. No nystagmus. Slight enlargement of glands in groin.

March 23. At 10 A.M., Dr. Castellani drew off about 40 c.c. cerebro-spinal fluid. Microscopically this fluid is teeming with cocci, single, in pairs, and in short chains. 20 c.c. of the fluid was centrifuged 15 minutes. The sediment contained many trypanosomes. The boy died at 3 P.M.

The following chart represents the course of the disease:—

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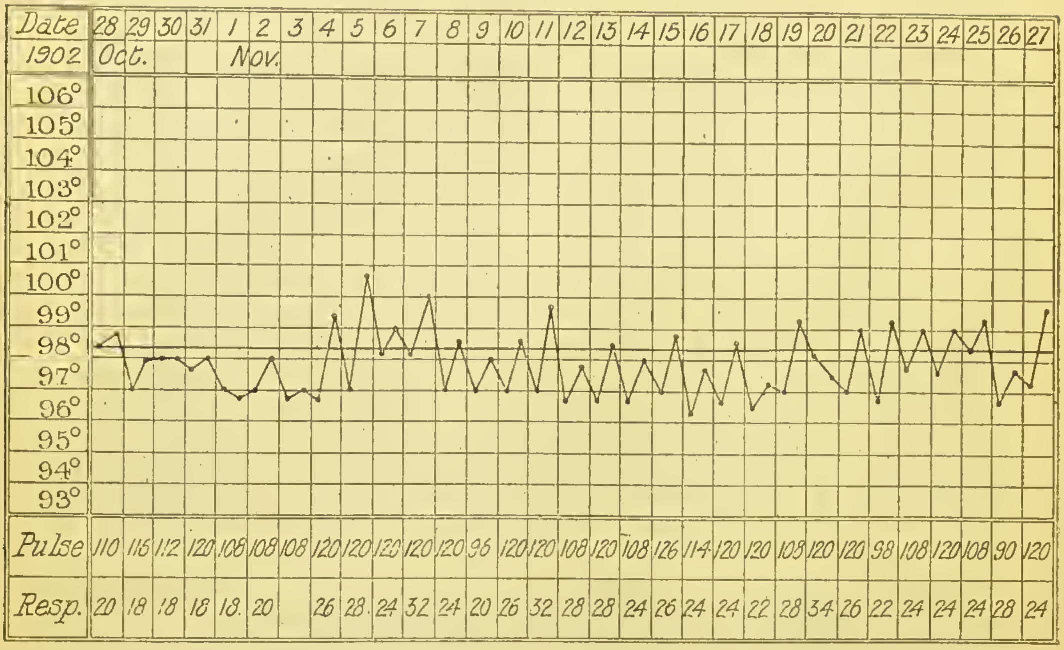
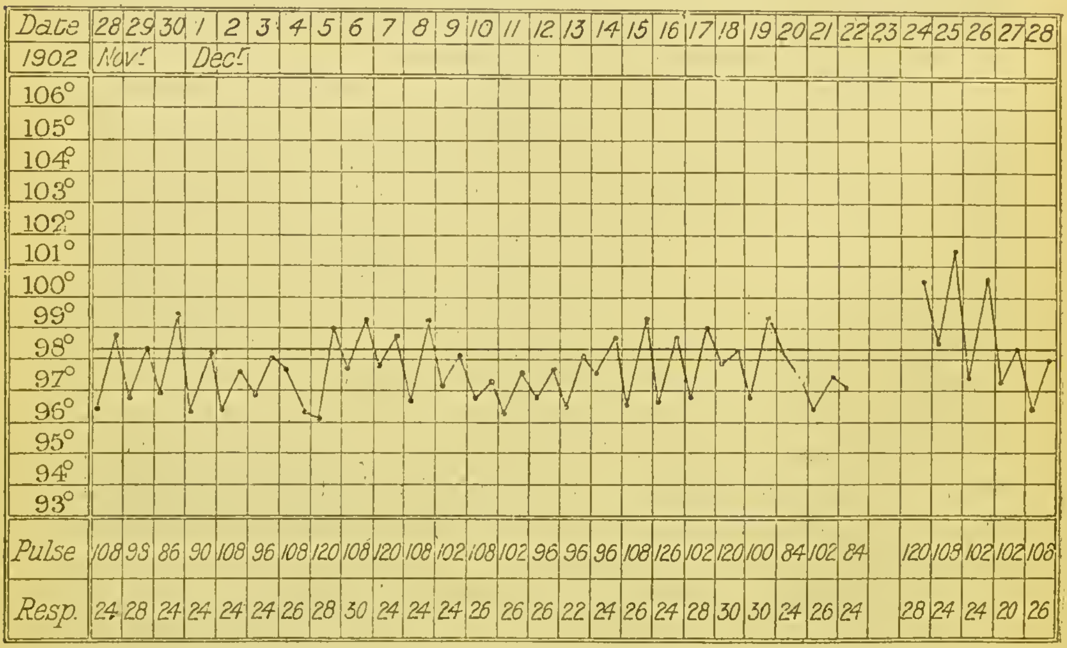
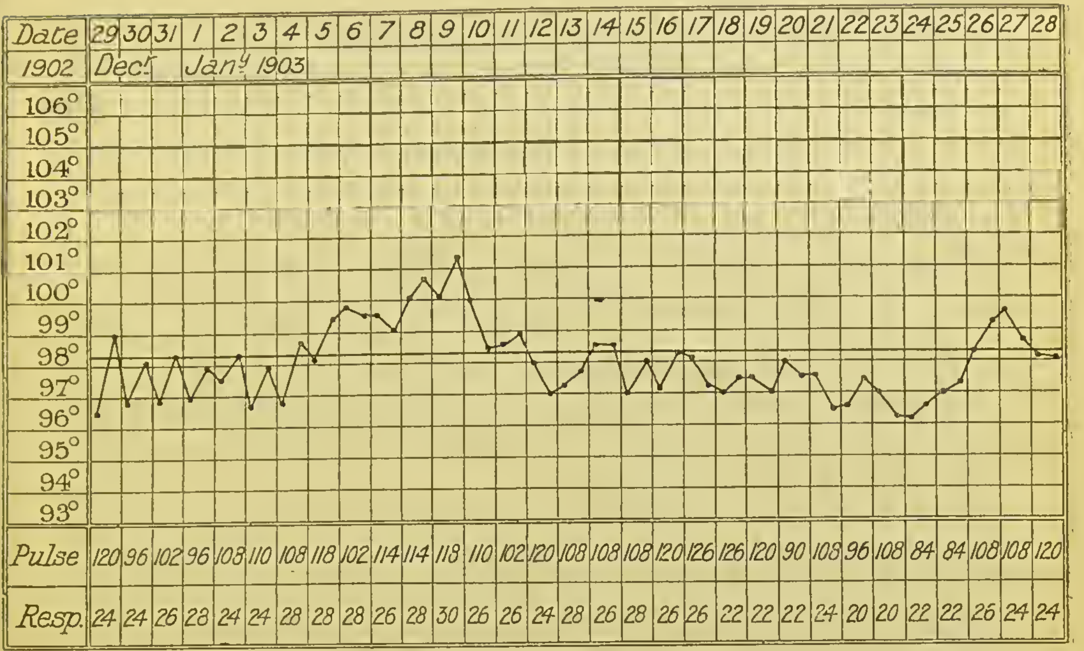
* [Some discrepancies may be observed between the charts and the clinical notes. It was deemed desirable not to defer the publication until these could be corrected by the authors.—Sec. R.S.]

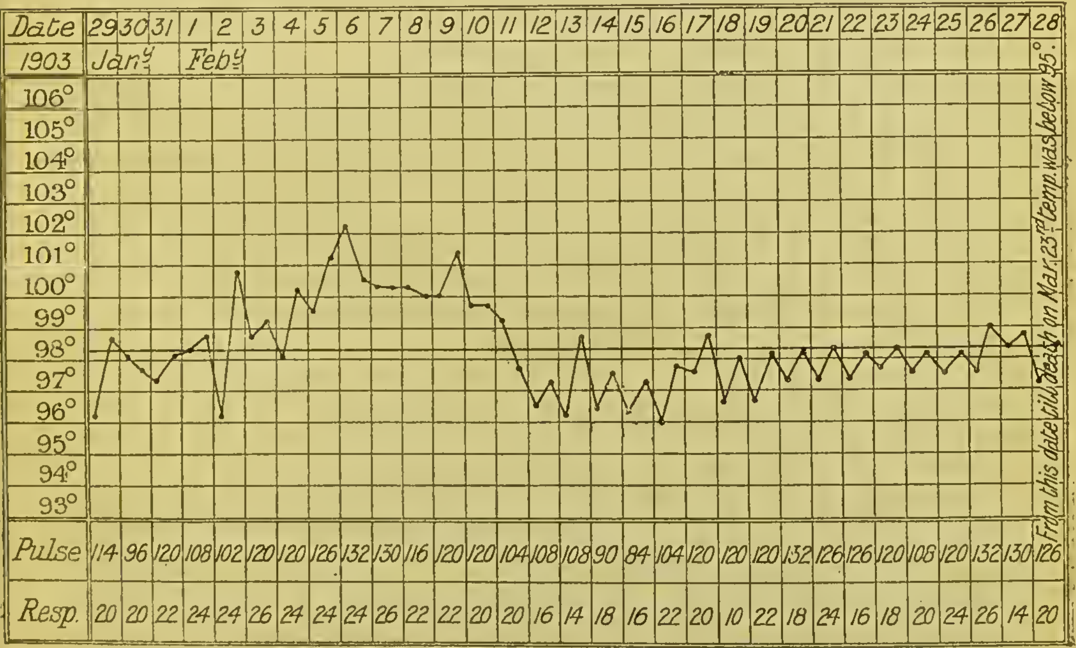


| Date | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1902 | Oct. | | | | Nov. | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 110 | 116 | 112 | 120 | 108 | 108 | 108 | 120 | 120 | 120 | 120 | 120 | 96 | 120 | 120 | 108 | 120 | 108 | 126 | 114 | 120 | 120 | 108 | 120 | 120 | 98 | 108 | 120 | 108 | 90 | 120 |
| Resp. | 20 | 18 | 18 | 18 | 18. | 20 | | 26 | 28 | 24 | 32 | 24 | 20 | 26 | 32 | 28 | 28 | 24 | 26 | 24 | 24 | 22 | 28 | 34 | 26 | 22 | 24 | 24 | 24 | 28 | 24 |



| Date | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1902 | Novr | | | Decr | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 108 | 98 | 86 | 90 | 108 | 96 | 108 | 120 | 108 | 120 | 108 | 102 | 108 | 102 | 96 | 96 | 96 | 108 | 126 | 102 | 120 | 100 | 84 | 102 | 84 | | 120 | 108 | 102 | 102 | 108 |
| Resp. | 24 | 28 | 24 | 24 | 24 | 24 | 26 | 28 | 30 | 24 | 24 | 24 | 26 | 26 | 26 | 22 | 24 | 26 | 24 | 28 | 30 | 30 | 24 | 26 | 24 | | 28 | 24 | 24 | 20 | 26 |





From this date till death on Mar. 23rd temp. was below 95°.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903.<br>Mar. 23.... | | | | – | + |

*Post-mortem* 2 hours after death.

Rigor mortis, absent. The body is that of an emaciated child about 8 to 10 years of age. There are no bed sores, but his feet are scabbed in places evidently due to jiggers.

On making an incision from the chin to the pubic region and reflecting the parietes, no fluid is found in the peritoneal cavity. The spleen is enlarged, remaining viscera appear healthy. On opening the chest cavity, no fluid is found in the pleural or pericardial cavities.

*Heart.*—Is flabby, the muscular tissue pale, otherwise normal.

*Lungs.*—Both lungs are perfectly healthy.

*Liver.*—Is pale, slightly fatty, otherwise healthy.

*Spleen.*—Is the usual malarial spleen, deeply pigmented, colour dark chocolate, firm, end fibrous to the touch.

*Stomach and Intestines.*—Externally normal.

*Brain.*—On removing the calvarium, the dura mater is non-adherent, smooth, pale in colour and fairly normal in appearance. The longitudinal sinus is filled with clotted blood. On cutting through the dura mater and reflecting it, the brain surface presents a highly diseased appearance. The dura mater is not adherent to the brain. The surface of the brain is in a condition of purulent meningitism. The small vessels are intensely congested. The convolutions are dusky red in colour or rather dotted with intense crimson on a yellowish ground (Plate 5).

Between the convolutions the sub-arachnoid fluid is in excess, is greenish-yellow in colour and evidently purulent.

Parts of the brain and spinal cord were put aside for microscopical examination. The fluid from the lateral ventricles is not purulent, but merely turbid, not from the presence of white blood corpuscles, but probably from the presence of innumerable cocci. This fluid also contains trypanosomes.

*Remarks.*—In our experience these acute inflammatory cases are not usual. In this case it is evidently due to streptococcal invasion.

This appearance is not typical of sleeping sickness; as a rule, there are no signs of acute inflammation, but merely a flattening of the convolutions, an injection of vessels, and an excess of sub-arachnoid fluid.

In Kaperi's case the cerebro-spinal fluid and blood were teeming with streptococci which would probably account for the acute inflammatory condition.

### CASE 35.—Fatoma (Female). Age 18 years. District, Usoga.

January 7, 1903. Admitted to hospital. States she had been ill for four months before coming into hospital. No relations had died of sleeping sickness. Has had syphilis.

April 7. *General Condition.*—Nutrition, flabby. Lips cracked. Gait weak and uncertain. Intelligence dull. Expression of face nothing noteworty. No œdema. Lymphatic glands generally enlarged.

*Nervous System.*—Intelligence dull. Sleeps much during the day. Speech indistinct and weak. Tongue furred, moist. No tremor of tongue, lips, or hands. Knee-jerks present. Ankle clonus absent.

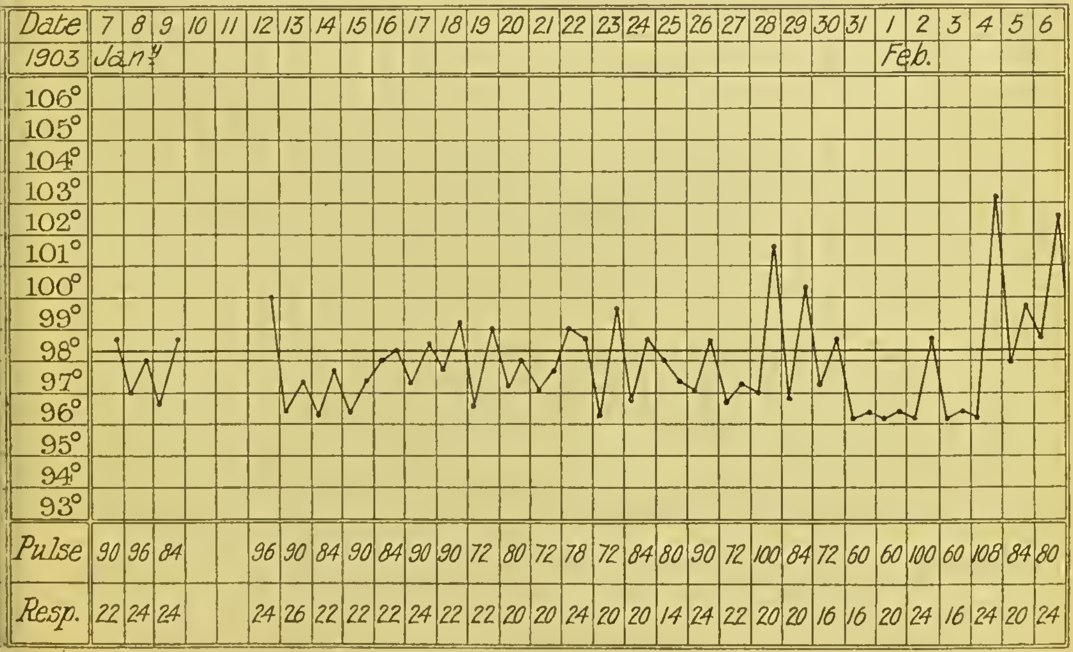
*Alimentary System.*—Appetite good. Liver and spleen not enlarged.

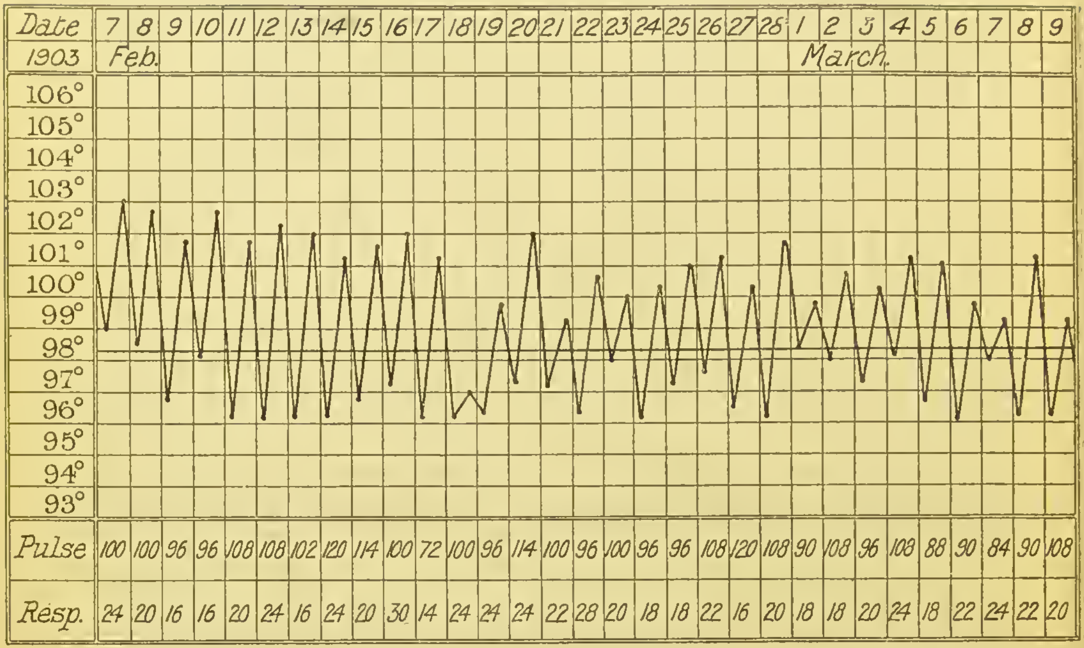
*Respiratory System.*—She has a cough. No physical signs of disease.

*Cutaneous System.*—There is an eruption of psoriasis over the trunk and extremities, probably syphilitic.

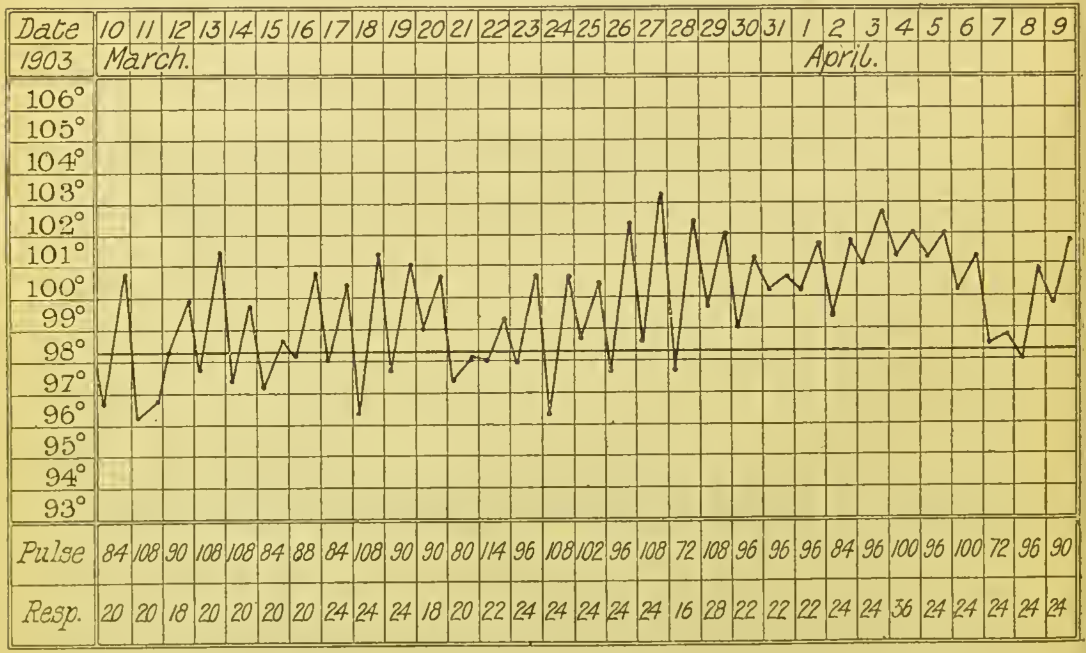
May 22. Died.

The following chart represents the course of the disease :—

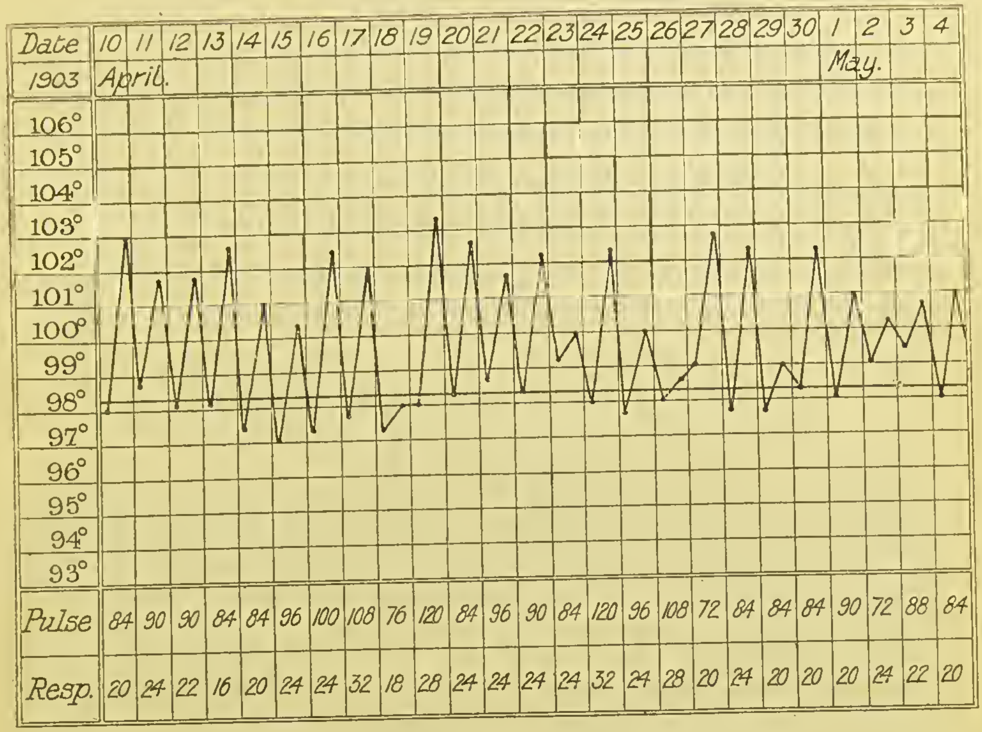


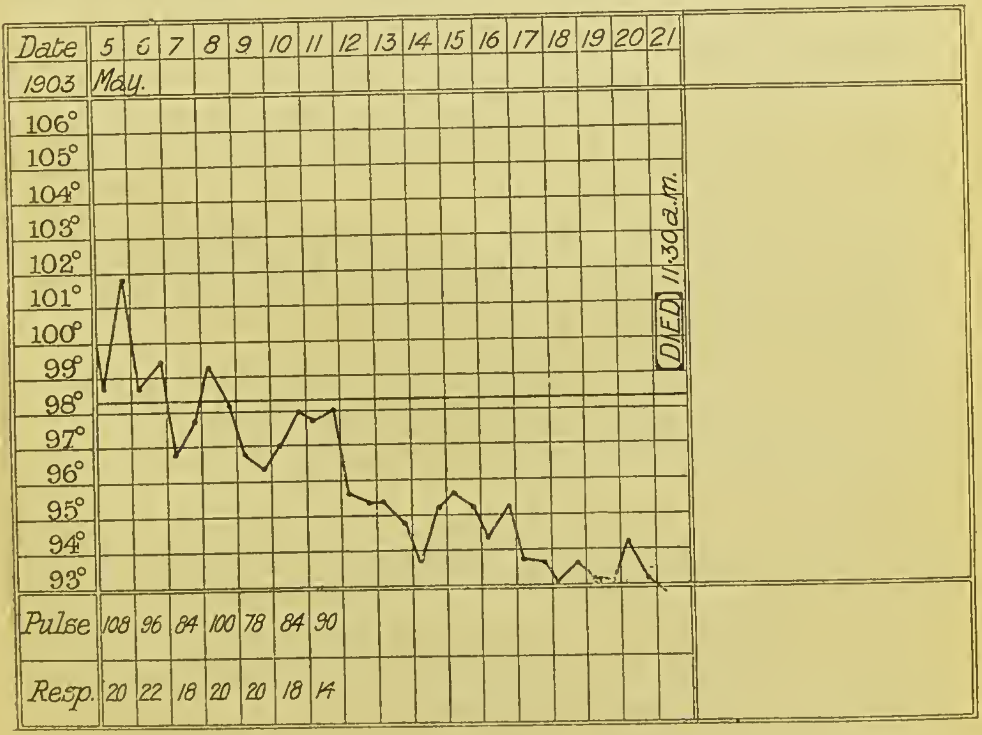


| Date | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | Feb. | | | | | | | | | | | | | | | | | | | | | | March. | | | | | | | | |
| Pulse | 100 | 100 | 96 | 96 | 108 | 108 | 102 | 120 | 114 | 100 | 72 | 100 | 96 | 114 | 100 | 96 | 100 | 96 | 96 | 108 | 120 | 108 | 90 | 108 | 96 | 108 | 88 | 90 | 84 | 90 | 108 |
| Resp. | 24 | 20 | 16 | 16 | 20 | 24 | 16 | 24 | 20 | 30 | 14 | 24 | 24 | 24 | 22 | 28 | 20 | 18 | 18 | 22 | 16 | 20 | 18 | 18 | 20 | 24 | 18 | 22 | 24 | 22 | 20 |



| Date | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | March. | | | | | | | | | | | | | | | | | | | | | | April. | | | | | | | | |
| Pulse | 84 | 108 | 90 | 108 | 108 | 84 | 88 | 84 | 108 | 90 | 90 | 80 | 114 | 96 | 108 | 102 | 96 | 108 | 72 | 108 | 96 | 96 | 96 | 84 | 96 | 100 | 96 | 100 | 72 | 96 | 90 |
| Resp. | 20 | 20 | 18 | 20 | 20 | 20 | 20 | 24 | 24 | 24 | 18 | 20 | 22 | 24 | 24 | 24 | 24 | 24 | 16 | 28 | 22 | 22 | 22 | 24 | 24 | 36 | 24 | 24 | 24 | 24 | 24 |


Date
1903
10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4
April.
May.
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
94°
93°
Pulse
84 90 90 84 84 96 100 108 76 120 84 96 90 84 120 96 108 72 84 84 84 90 72 88 84
Resp.
20 24 22 16 20 24 24 32 18 28 24 24 24 24 32 24 28 20 24 20 20 20 24 22 20


Date
1903
5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
May.
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
94°
93°
DIED 11.30 a.m.
Pulse
108 96 84 100 78 84 90
Resp.
20 22 18 20 20 18 14

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 26.... | | | | | – |
| Apr. 1.... | | | | | + |
| ,, 22.... | | – | | | |

May 22. *Post-mortem* 2½ hours after death.

Rigor mortis not yet present. The body is that of a young woman fairly well nourished, scaly eruption over body, and commencing bedsore on the hips. There is slight enlargement of the superficial lymphatic glands.

Body opened in the usual way. About 3 oz. clear straw-coloured fluid found in the pericardial cavity, none in pleural or peritoneal cavities. The lymphatic glands lying behind the manubrium and over the trachea were found much enlarged, the mass of them as large as a Tangerine orange. From this mass other glands extended up into the neck beneath both clavicles and downwards towards the root of the lungs. On section the glands were either caseating or full of pus.

*Heart.*—Weighed 6 ozs. Nothing abnormal present. Muscle substance firm and of good colour.

*Lungs.*—Right, weighed 13½ ozs. The lower part of the upper lobe was solid, and on section showed beads of pus in many places, other parts of this area looked brownish and gelatinous. The lower lobe was congested, but no pus was present.

Left, 6½ ozs. in weight. Congested, but not suppurating anywhere. Near the lower parts there was a small hard nodule, gelatinous on section. The glands in the hilum of the lungs were either caseating or suppurating.

*Liver.*—The liver is very big, weight 4 lbs. Very firm and tough in consistence. Nothing abnormal on section except some increase of the fibrous trabeculæ.

*Spleen.*—Weight 8 lbs. Enlarged. Almost universally adherent. On section the surface is dotted with small yellow necrotic areas varying in size from a pin's head to a small pea.

*Kidneys.*—Each weighed 3½ lbs. Cortex congested, capsule strips readily.

*Glands.*—The retroperitoneal glands are greatly enlarged, forming a mass in front of the spine, surrounding the mesenteric vessels and pancreas, and forming a large tumour projecting in the small curvature of the stomach. On removal the mass of glands weighed 1½ lbs. On section most of the glands were converted into collections of pus, some of them being inflamed and in a commencing stage of disintegration. The inguinal glands were slightly enlarged and no signs of pus present in them.

*Brain.*—Much fluid escaped on removing the skull cap. On removing the dura mater, there was found increase of the sub-arachnoid fluid with the opaque ground-glass appearance of the sulci. Some congestion of vessels on the surface of the brain. Substance tough, otherwise normal.

### CASE 19.—Noubi. Female. Age 30 years.

February 23, 1903. Admitted to hospital.

April 7. She has a heavy look, walks weakly and with hesitation. She talks indistinctly and weakly as if she had a pebble in her mouth. She states that three brothers and four sisters all died of sleeping sickness. She has lately come from Bugonga near Entebbe, but came from Busoga some time ago.

April 18. *General Condition.*—This patient can neither speak nor walk. She is conscious and tries to speak, and has an anxious staring expression. She is well nourished. There is no œdema. The conjunctivæ are pale and anæmic. There are no trophic changes. The lymphatic glands are enlarged to the size of beans. Her breathing is quiet and shallow, and her pulse is almost imperceptible, there are no cutaneous eruptions.

*Nervous System.*—She is conscious and tries to answer questions, but cannot. She lies all day with her eyes open, absolutely torpid. There is slight lateral nystagmus, the pupils are equal in size and she can see. There is no reaction to light. No facial palsy, and she cannot put out her tongue. She is sensible to touch. There is some twitching of the muscles. Knee-jerk is absent; no ankle clonus.

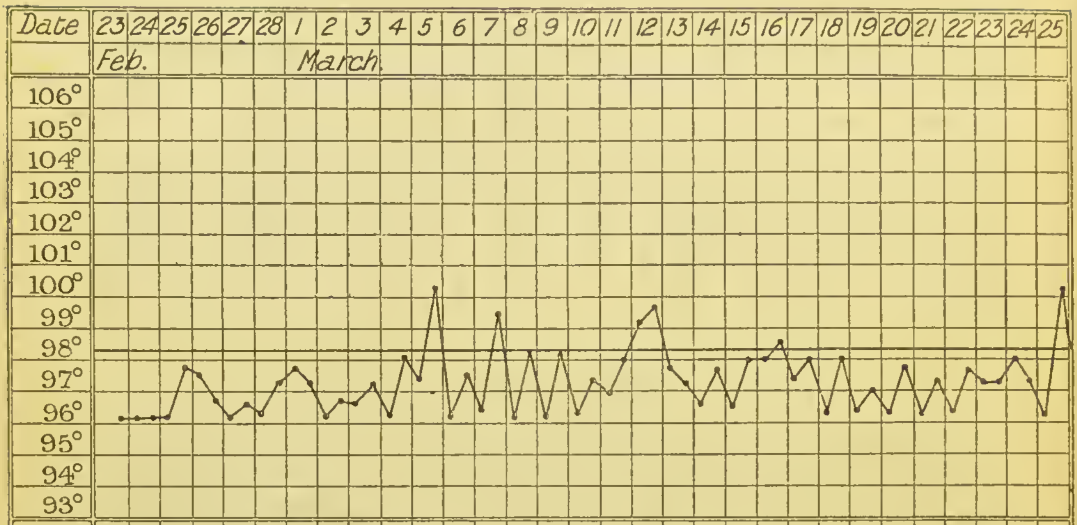
*Alimentary System.*—Has not been able to eat since yesterday. The spleen is enlarged and hard. The liver is not enlarged. Fæces and urine passed involuntarily.

*Circulatory System.*—Heart sounds normal, but weak.

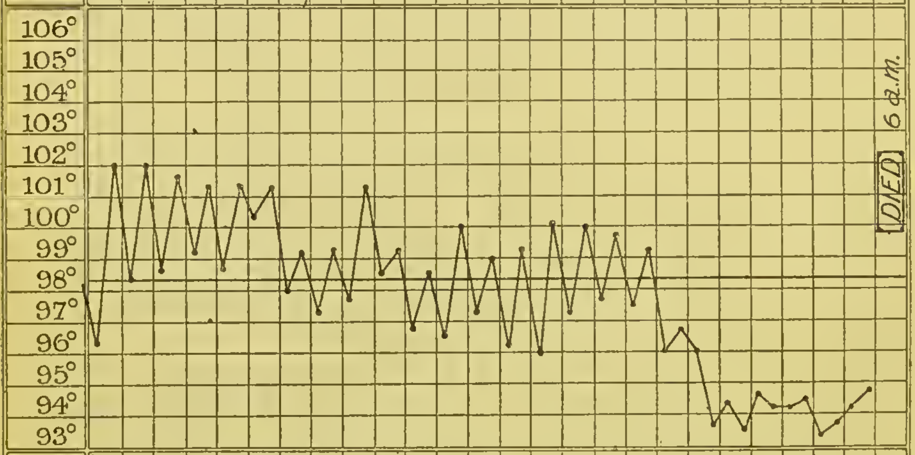
*Skin.*—Is harsh, dry, rough, and scaly on the legs.

April 20. Died at 6 A.M.

The following chart represents the course of the disease :—



| Date | 23 | 24 | 25 | 26 | 27 | 28 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  | Feb. |  |  |  |  |  | March |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Pulse | 80 | 96 | 96 | 104 | 108 | 84 | 72 | 96 | 108 | 108 | 100 | 108 | 96 | 100 | 90 | 90 | 100 | 100 | 100 | 88 | 108 | 108 | 88 | 100 | 80 | 80 | 90 | 96 | 100 | 96 | 100 |
| Resp. | 24 | 24 | 18 | 28 | 24 | 20 | 16 | 16 | 24 | 24 | 24 | 24 | 24 | 22 | 20 | 24 | 22 | 24 | 24 | 24 | 18 | 24 | 16 | 24 | 20 | 20 | 20 | 18 | 22 | 24 | 24 |



| Date | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  | March. |  |  |  |  |  | April. |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Pulse | 108 | 90 | 72 | 114 | 100 | 100 | 104 | 108 | 140 | 108 | 84 | 84 | 96 | 84 | 108 | 108 | 108 | 108 | 96 | 108 | 100 | 96 | 96 | 72 |  |  |
| Resp. | 18 | 20 | 12 | 20 | 26 | 18 | 20 | 20 | 36 | 24 | 20 | 14 | 24 | 20 | 24 | 26 | 20 | 16 | 16 | 36 | 20 | 24 | 32 | 24 |  |  |

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 24.... | | | | | — |
| Apr. 13.... | + | | | | + |
| „ 17.... | + | — | — | | |
| „ 19.... | · | | | | |
| „ 19.... | | | — | | |

April 20. *Post-mortem*, 9 A.M., 3 hours after death.

The body is that of a well-nourished young woman. Rigor mortis is absent. There are no bed sores. The pupils are equal and normal in size. On making an incision from the chin to the pubic region, and reflecting the parietes, there is no fluid found in the peritoneal or pleural cavities.

The pericardium contains an excess of turbid fluid containing flakes of lymph. The pericardium, visceral and parietal, is in an acutely inflamed condition. Its colour is dusky red, and there are flakes of recent lymph over the surface.

*Heart.*—There is an excess of fat about the base. The whole surface is dark red in colour, especially anteriorly. On section the muscle is pale, otherwise the valves, etc., are normal. No petechiæ on the endocardium.

*Lungs.*—Right. The glands at the base are enlarged (size of a bean). On section the lower lobe is congested, but no signs of pneumonia. Left lung is normal.

*Spleen.*—Is much enlarged (8 by 5 inches), and must weigh a couple of pounds. The capsule is partly adherent to the surrounding structures. On section the substance is rather tough, with little or no signs of pigmentation and dark reddish-brown in colour. Smears stained by Leishman's stain showed no malarial parasites.

*Liver.*—Is not enlarged. There are a few adhesions of the right lobe to the diaphragm. On section the liver tissue is slightly pigmented and fatty. The texture is somewhat tough.

*Kidneys.*—Normal.

*Intestines.*—The mesenteric glands are enlarged. There are some round worms, and, 6 inches below pylorus, about 30 ankylostomata Nothing else noteworthy.

*Brain.*—The dura mater is normal. The convolutions of the surface of the brain are flattened, the sulci filled with opaque looking subarachnoid fluid. The vessels are injected, otherwise nothing abnormal.

*Remarks.*—This is an ordinary case of sleeping sickness. The anæmia may have been helped by the ankylostomata. The pericarditis is an uncommon feature.

### Case 23.—Budara (Male). Age 22 years. Occupation, Kitchen Boy. District, near Kampala.

Admitted February 26, 1903, to hospital complaining of weakness. He has a dull look and tremors of the tongue and hands. States he has been ill about a month.

April 8. *General Condition.*—He is thin and poorly nourished. His gait is feeble. He is very slow and lethargic in his movements, and his face is dull and expressionless. There is no œdema of any part. The colour of his mucous membranes is normal. Lymphatic glands generally enlarged (the size of beans). He does not complain of any pain, and there are no cutaneous eruptions.

*Nervous System.*—His intelligence is dull and he sleeps a good deal. His speech is slow, but distinct, and very weak, not being raised above a whisper. His vision and the movements of his eyes appear to be normal. He has marked tremor of the tongue, but not of the lips or hands. His knee-jerk is absent, and ankle clonus is not present.

*Alimentary System.*—His tongue is moist and slightly furred, and his lips are dry. Neither liver nor spleen is enlarged.

*Circulatory System.*—The heart's impulse is not visible or palpable. On auscultation the sounds are weak, but normal. The pulse is 70 per minute, with a low tension, small in size, easily compressible and rhythm regular.

*Respiratory System.*—Nothing noteworthy.

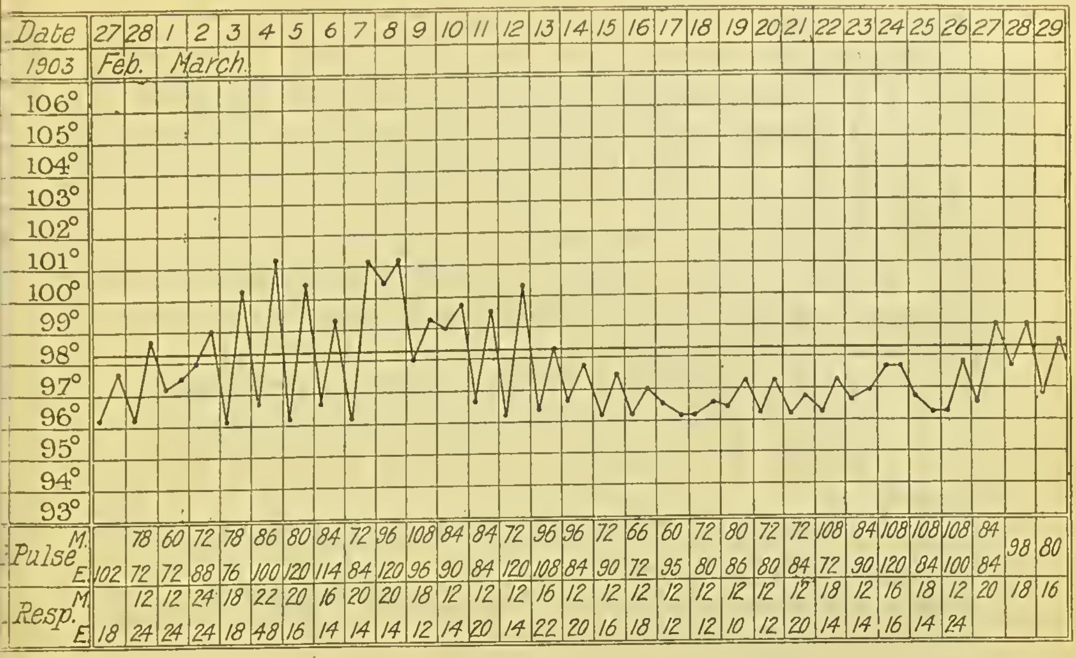
*Urinary System.*—Nothing noted.

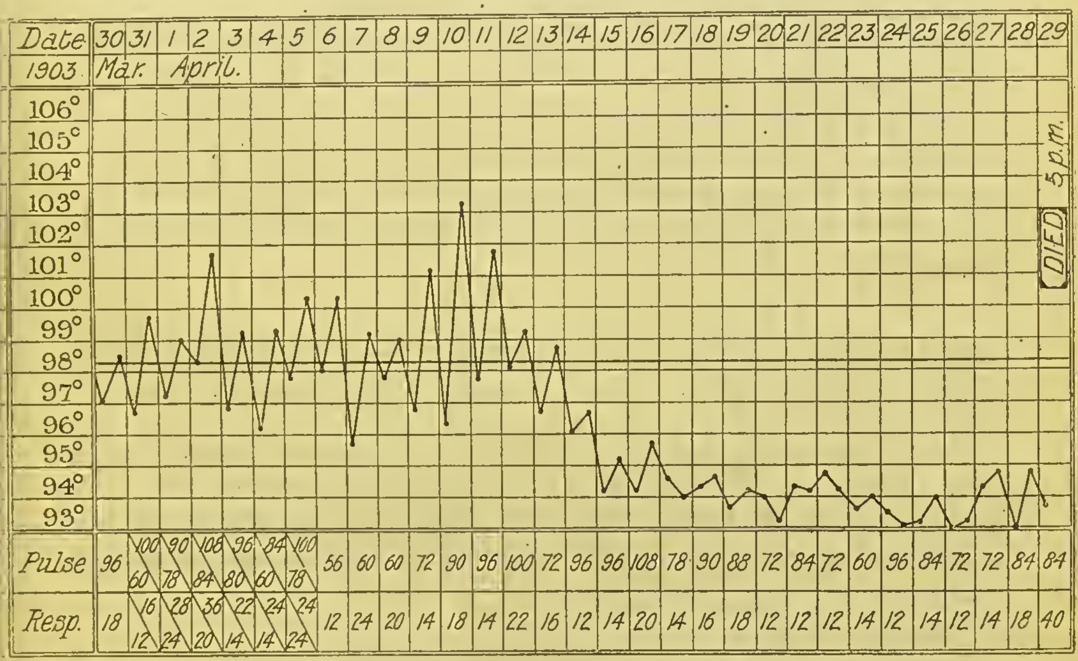
*Skin.*—Over the legs and forearms it is scaly, rough, and dry.

The following chart (p. 51) represents the course of the disease.

The following table shows the absence or presence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 26.... | | | | — | + |
| Apr. 8.... | — | — | — | | |
| ,, 18.... | + | — | + | | |
| ,, 22.... | | — | | | |





April 25, 1903. During the past few days the patient's temperature has been constantly below 95° F. and his body feels cold to the hand. He is becoming much more apathetic, and his eyes are half closed.

April 27, 1903. Patient is now practically dying. His eyes are nearly closed and he does not respond when spoken to. He takes milk with difficulty.

April 29, 1903. He had an epileptiform seizure at noon. Blood taken for examination was very dark and venous. Died at 5 P.M.

April 30, 1903. *Post-mortem* 16½ hours after death.

Rigor mortis is present. There are no bed sores. The pupils are equal. The body is that of a thin, but well nourished, man of 20.

*General.*—On opening the body no fluid is found in the peritoneal or pleural cavities. There is a small amount of clear straw-coloured fluid in the pericardium.

*Heart.*—Weight 8 oz. On the anterior surface of the heart over the right ventricle there were several well-marked petechiæ, and this condition was also found in the endocardium, especially in the left ventricle. Otherwise the heart is normal.

*Lungs.*—Right weighed 7 oz. On section it was congested.
Left weighed 7 oz. On section it was fairly normal.

*Liver.*—Weight 3 lb. 12 oz. On section there was some fatty degeneration.

*Spleen.*—Weight 8 oz. On section it was slightly pigmented, and there is an excess of fibrous tissue.

*Kidneys.*—Both weighed 2½ oz. The capsules stripped off readily.

*Pancreas.*—Nothing noteworthy.

*Intestines.*—The mesenteric glands are all enlarged.

*Brain.*—This is an ordinary typical sleeping-sickness brain with flattened convolutions, excess of sub-arachnoid fluid giving the surface an opaque, glassy appearance. There is an excess of fluid in the ventricles, otherwise nothing abnormal can be seen by the naked eye.

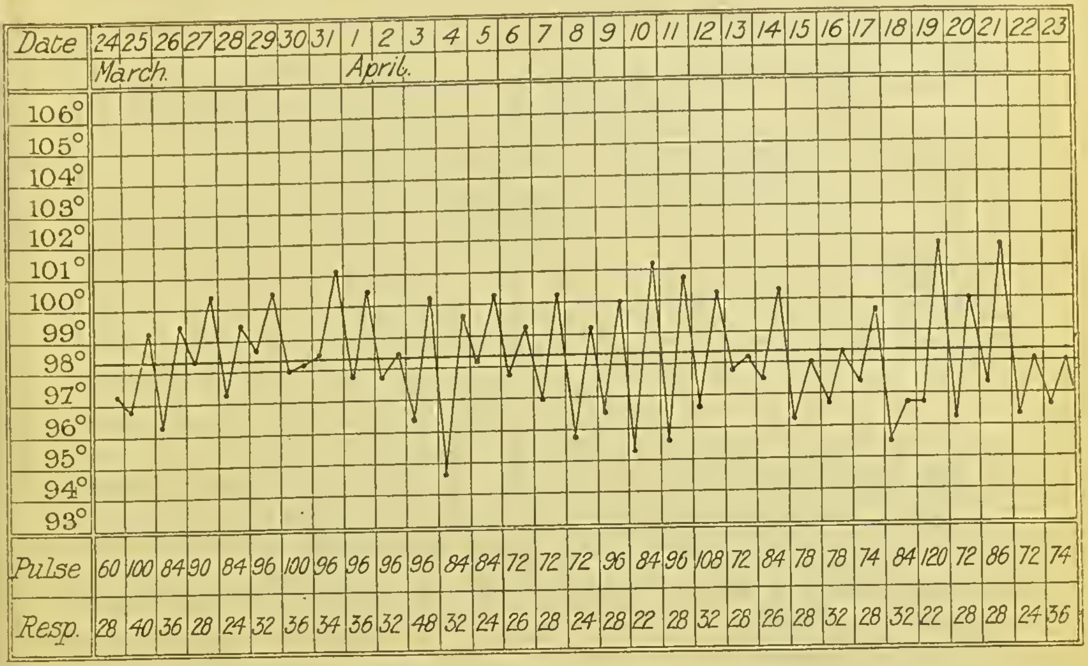
*Remarks.*—This is an ordinary uncomplicated case of sleeping sickness. During the last day or two of life he had convulsive seizures, in one of which he died.

CASE 18.—Kavera (Male). Age 52 years. Control. District, Bussi. Occupation, Fisherman. Food, Bananas, Fish, and Sweet Potatoes.

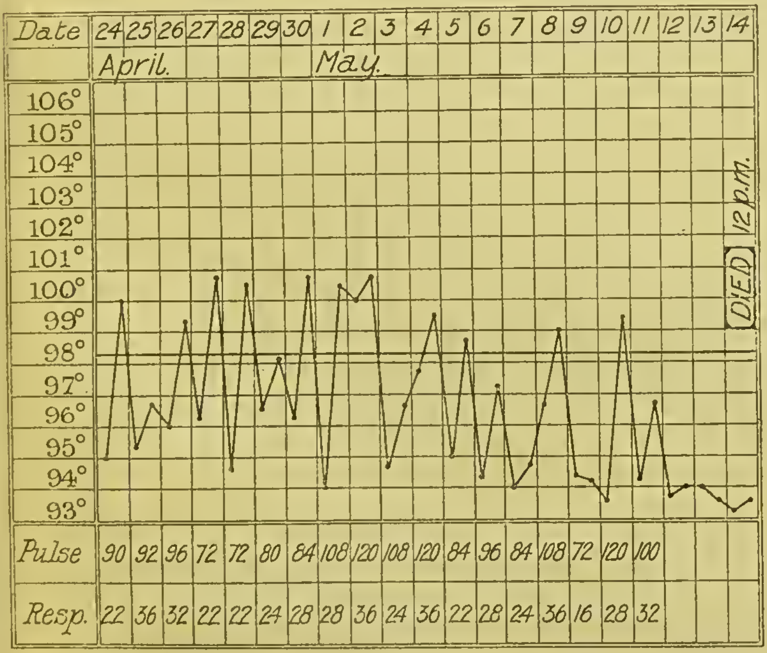
March 24, 1903. Admitted to hospital as a case of sleeping sickness. He is an old man, bent double with rheumatism, and has a large ischio-rectal abscess. States he has been ill 3 months, the first symptoms being pain in the back. There had been no cases of sleeping sickness in the same house.

May 13. Died.

The following chart represents the course of the disease:—



| Date | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | March. | | | | | | | | April. | | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 60 | 100 | 84 | 90 | 84 | 96 | 100 | 96 | 96 | 96 | 96 | 84 | 84 | 72 | 72 | 72 | 96 | 84 | 96 | 108 | 72 | 84 | 78 | 78 | 74 | 84 | 120 | 72 | 86 | 72 | 74 |
| Resp. | 28 | 40 | 36 | 28 | 24 | 32 | 36 | 34 | 36 | 32 | 48 | 32 | 24 | 26 | 28 | 24 | 28 | 22 | 28 | 32 | 28 | 26 | 28 | 32 | 28 | 32 | 22 | 28 | 28 | 24 | 36 |



| Date | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | April. | | | | | | | May. | | | | | | | | | | | | | |
| Pulse | 90 | 92 | 96 | 72 | 72 | 80 | 84 | 108 | 120 | 108 | 120 | 84 | 96 | 84 | 108 | 72 | 120 | 100 | | | |
| Resp. | 22 | 36 | 32 | 22 | 22 | 24 | 28 | 28 | 36 | 24 | 36 | 22 | 28 | 24 | 36 | 16 | 28 | 32 | | | |

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 6.... | | | | | — |
| „ 22.... | | — | | | |

May 14. *Post-mortem* 9 hours after death.

Rigor mortis passing off. There is a superficial bed sore over sacrum. The body is extremely emaciated, the right testicle enlarged, and there is a large fistula in the ischio-rectal region.

*Heart.*—The wall of the right ventricle is thinned; the muscular tissue of the left dark in colour, tough and fibrous.

*Lungs.*—Healthy.

*Liver.*—Weight 2 lb. 5 oz. Small on section, slate-coloured and tough.

*Spleen.*—Universally adherent. Weighed 4 oz. There is a small tumour, the size of a kidney bean in the substance, probably secondary.

*Kidneys.*—Left weighs 3 oz. Capsule strips readily, substance normal.
Right weighs 2½ oz. Capsule strips readily, substance normal.

*Alimentary System.*—Lymphatic glands enlarged. A small tumour the size of a walnut attached to mesentery. Abscess in right testicle, probably communicating with fistulous opening near anus.

*Remarks.*—There was no reason at any time to suppose that this was a case of sleeping sickness. His case is given as a control. He also, like Case 26, Zenabu, was for 2 months in close contact with sleeping sickness cases without contracting the disease.

### CASE 26.—Zenabu (Female). Age 18 years. Control. No appreciable disease. District, Budu. Food, Sweet Potatoes.

February 27, 1903. Admitted to hospital as a case of sleeping sickness. States the first symptom was headache; has never been drowsy.

March 30. Case taken. There was nothing definite, headache and amenorrhœa. She has been ill 3 months.

*General Condition.*—Development and nutrition good. Gait normal, but she lies about a good deal. She is fairly intelligent, but the expression of her face is somewhat dull. There is no œdematous swelling in any part of her body. She is somewhat anæmic. Her lymphatic glands are generally enlarged (size of peas). She complains of pain in the back of her neck. Her breathing is normal, 16 per minute. Her pulse 96 per minute and her temperature 98·8. There is a papulo-vesicular eruption, evidently due to itch, in the dorsum of both hands and on arms and forearms.

*Nervous System.*—Her intelligence and memory seem normal, her sleep and speech also normal. Her eyes present nothing noteworthy. She has no tremors of tongue, lips, or hands, her left knee-jerk is increased, the right is normal; no ankle clonus.

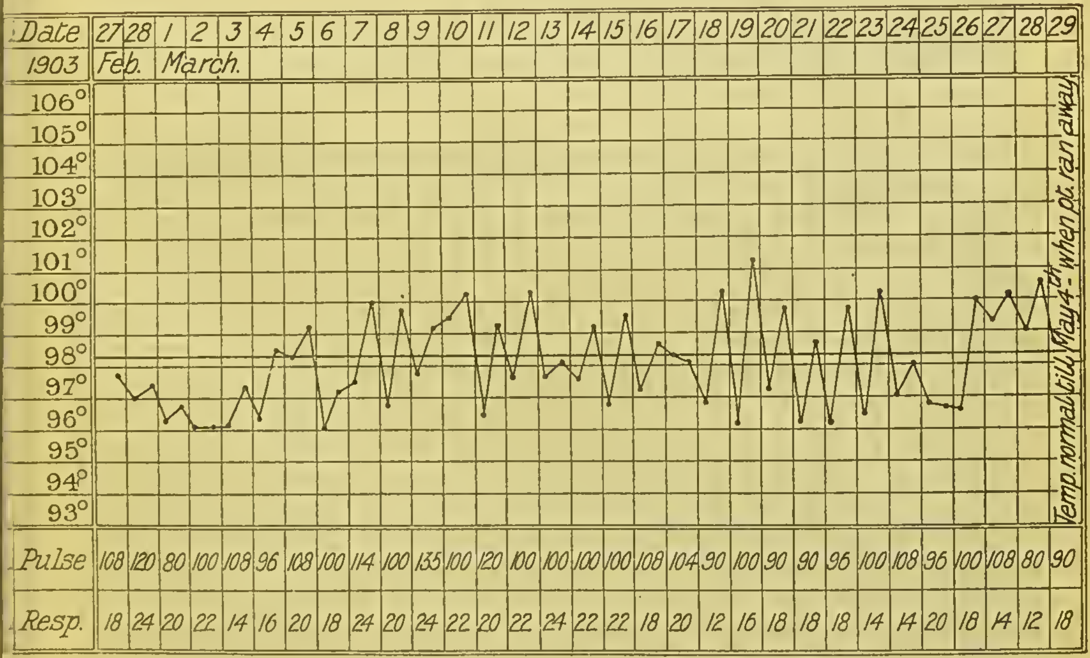
*Liver and Spleen.*—Not markedly enlarged.

*Heart.*—The heart impulse is feeble and is not easily felt, but the sounds are normal in character. The pulse has a rate of 96, a fair tension, small in size, normal in compressibility, and regular in rhythm.

*Eyes.*—On ophthalmoscopic examination her eyes were found to be normal.

May 4. Ran away.

The following chart represents the course of the disease :—



The temperature remained normal from March 29th to May 4th, when patient ran away.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 9.... | + | – | – | | |
| „ 13.... | | | | + | – |
| „ 22.... | | – | | | |

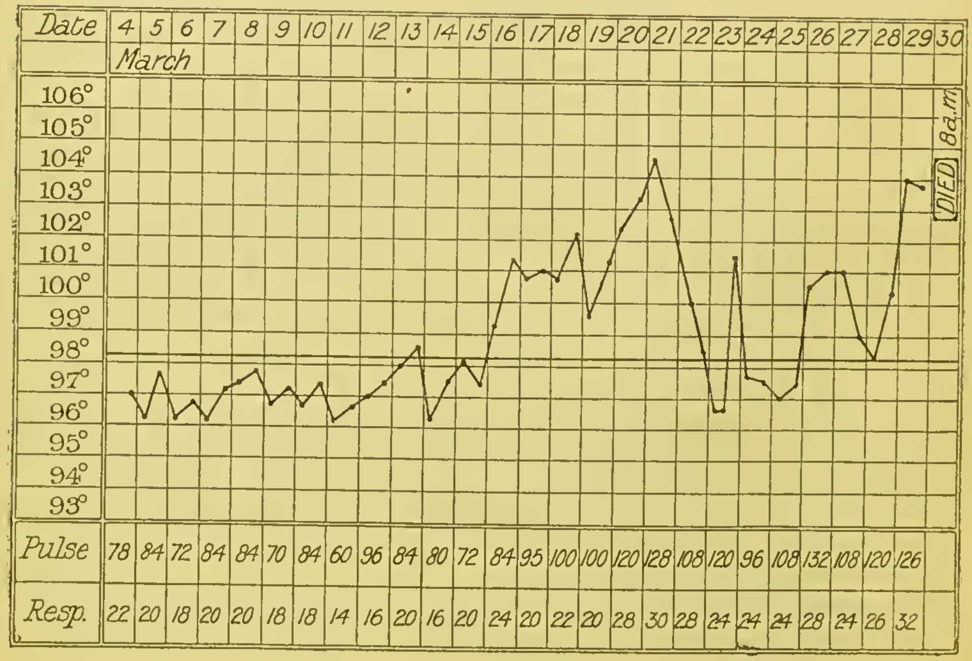
*Remarks.*—This is no case of sleeping sickness. It was intended to send her home in a few days. She was in the Sleeping Sickness Hospital for more than 2 months, in close contact with the patients, without being affected. This would go to show that lice and fleas do not convey the disease, as these insects were numerous in the hospital when we arrived here.

### Case 12.—Ibraham (Male). Age 20 years.

March 4, 1903. Admitted to hospital.

March 30. Died.

The following chart represents the course of the disease :—



*Post-mortem* 1½ hours after death.

Rigor mortis absent. There is a large bed sore over the sacrum. Lymphatic glands of the groin are enlarged. An abscess was found in the region of the third, fourth, and fifth lumbar vertebræ. Probably caused by the bed sore.

*Brain.*—Dura mater non-adherent, normal. The vessels on the surface of the brain are injected. Sub-arachnoid fluid in excess, giving a dull glassy œdematous look, similar to Case 11, but not quite so well marked.

*Heart.*—There is some yellow œdematous material about the base. The interior is normal. Aorta healthy.

*Lungs.*—Area of collapse at base of right, otherwise healthy.

*Liver.*—Large, somewhat pigmented, marked fatty degeneration.

*Spleen.*—Very large, pigmented, substance softened, almost diffluent.

*Kidneys.*—Normal. *Pancreas*, healthy.

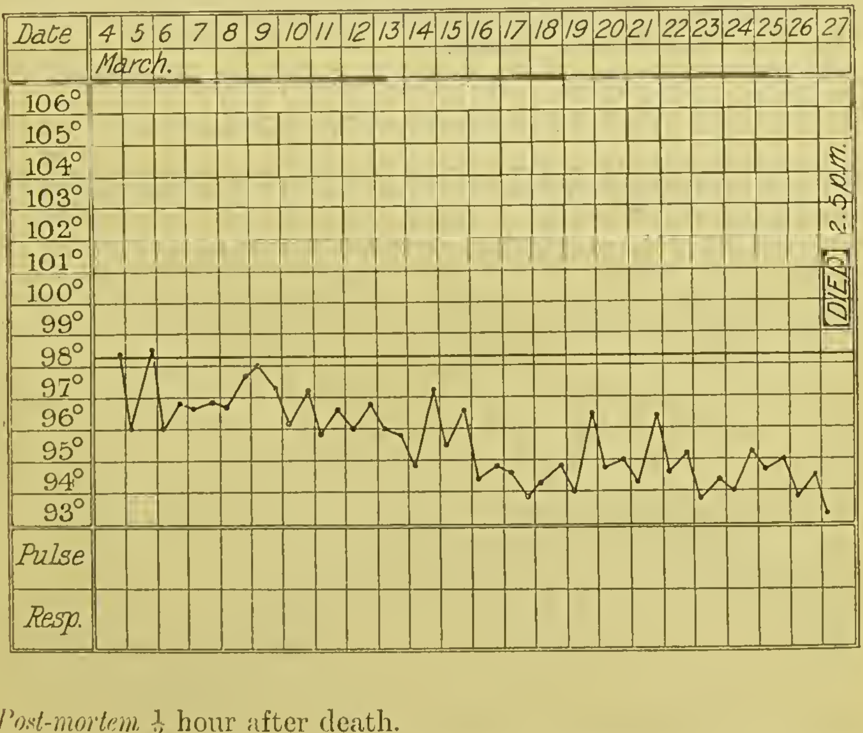
*Intestines.*—Mesenteric glands enlarged. No ankylostomata.

### CASE 10.—Ibsasara (Male). Age about 35.

March 5. Admitted to hospital.

March 27. Died at 2 P.M.

The following chart represents the temperature curve :—



*Post-mortem* ½ hour after death.

Rigor mortis absent. Large bed sore over sacrum. Body extremely emaciated, reduced to skin and bone. There was no excess of fluid in the pericardium, pleural or peritoneal cavities.

On removing the calvarium a large quantity of blood escaped, it not having as yet clotted. The dura mater was not adherent to the brain, nor did it appear in any way abnormal.

On reflecting the dura mater the brain was found to have an abnormal appearance. The small vessels on the surface were injected, the pia mater was opaque, and the sub-arachnoid fluid much in excess, giving the exposed surface of the brain a dull glassy appearance.

The organs generally were healthy.

*Spleen.*—Was normal in size, but deeply pigmented and tough.

*Liver.*—Was small, dark chocolate in colour, and somewhat firmer than usual.

*Heart.*—Was healthy.

*Lungs.*—Were fairly normal, except for a patch of congestion and œdema in the superior lobe of the right lung.

*Kidneys.*—Appeared healthy.

*Large Intestine.*—Was packed full of masses of hard fæces.

*Remarks.*—This case shows the tendency to obstinate constipation towards the end of the disease.

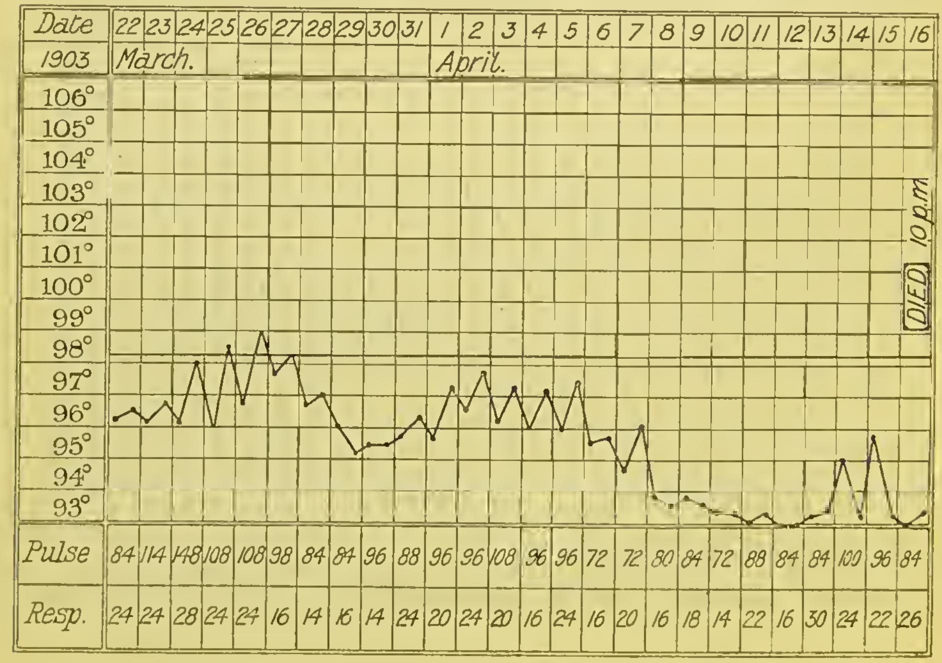
### CASE 39.—Zakibu (Male). Age 20.

March 22, 1903. Admitted to hospital. Patient has a tottering gait. His glands are enlarged, temperature normal, and no tremors.

April 14. General convulsions.

April 16. Died 10 P.M. Left facial palsy before death.

The following chart represents the course of the disease :—



The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903.<br>Apr. 6 ..... | | | | | + |

April 17. *Post-mortem*, 9.30 P.M., 11½ hours after death.

The body is that of a well-nourished young man. Rigor mortis is present. There is a superficial bed sore over the right hip, and a bulla over the front of left thigh. Right pupil somewhat larger than the left.

*Heart.*—Normal in size. Valves and muscular substance healthy, no petechiæ.

*Lungs.*—Right. Nearly the whole of the upper lobe and upper half of the lower lobe is consolidated. The middle lobe is normal. On section the above-named parts are found to be in a condition of red hepatisation.

Left. This lung is firmly adherent to the diaphragm by old adhesions. No fluid in either pleural cavity. The lower lobe is deeply congested.

*Spleen.*—Much enlarged and adherent to surrounding structures. On removing it, it is found to be distorted in shape, due to a mass of cicatricial tissue which has become calcareous, evidently the site of an old infarct. The substance is dark, pigmented, and tough.

*Liver.*—Large, capsule smooth. On section slightly pigmented, otherwise nothing noteworthy.

*Kidneys.*—Healthy. *Intestines.*—Healthy. Some worms in the upper part of small intestine.

*Scalp.*—A patch of œdematous yellow jelly-like material.

*Brain.*—On removing the calvarium the dura mater is found to be healthy in appearance.

The surface of the brain is injected and the sub-arachnoid fluid in excess and dull and opaque in character. There are no signs of active or acute meningitis. The substance of the brain appears healthy to the normal eye.

*Remarks.*—This case ended in pneumonia with a pneumococcus invasion of the blood.

CASE 40.—Matasa (Male). Age 24 years. District, Bussi. Occupation, Bark-cloth Maker. Food, Bananas and Sweet Potatoes, no Fish.

March 24. Admitted to hospital; facies dull, stares vacantly; gait somewhat weak; knee-jerks feeble; pulse fair, 100; tremor of lips and fingers. He states that no other cases of sleeping sickness had occurred in the same house. Had been ill about 2 months, the first symptoms being pain in the head and chest, then he slept too much.

April 15, 1903. *General Condition.*—He is well nourished, has a weak and staggering gait, is slow, and the expression of his face is dull and vacant. There are no œdematous swellings or trophic changes. The lymphatic glands are enlarged, especially in the inguinal and femoral regions (the size of beans). He complains of headache, his pulse is 102 standing up, temperature 99·8, and there are no cutaneous eruptions.

*Nervous System.*—His intelligence and memory are fair; he is always lying in bed in a drowsy condition. His speech is slow and mumbling. His eyes are normal. There is a marked tremor of tongue, lips, hands, and generally of the body. Sensibility to touch is normal. In regard to the reflexes, knee-jerk is present, ankle clonus absent.

*Alimentary System.*—His appetite is good, his tongue is moist and furred, his liver is not enlarged, his spleen is not palpable.

*Circulatory System.*—Heart sounds are normal. Pulse 132, tension low, size small, compressibility easy, rhythm slightly irregular.

*Respiratory System.*—Nothing abnormal.

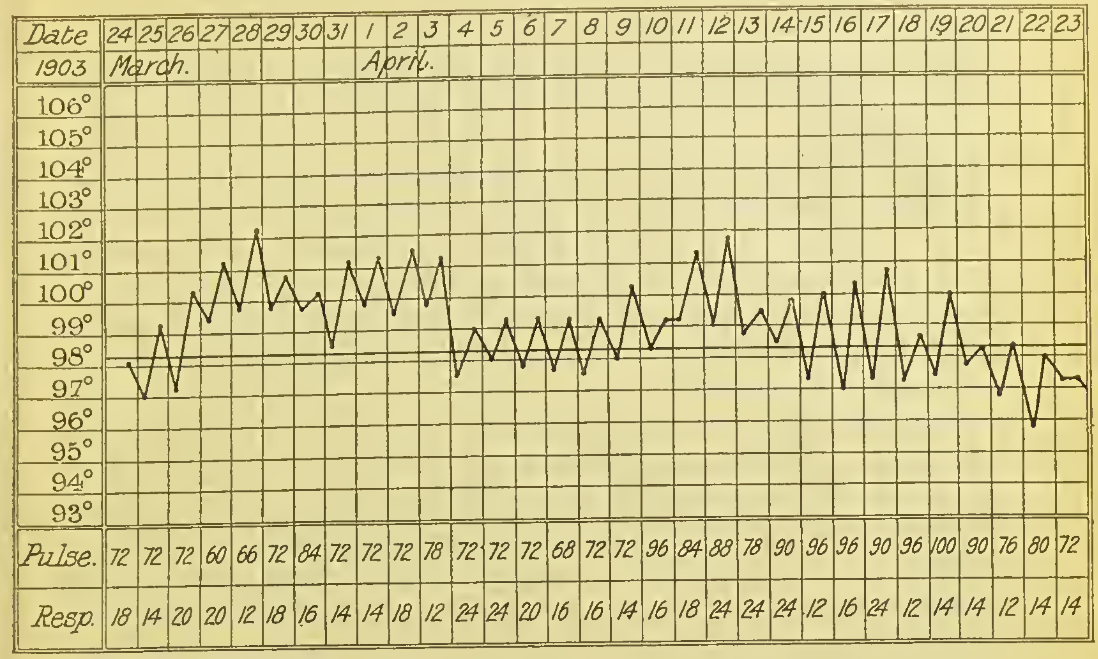
*Skin.*—The skin of the legs is scaly and dry.

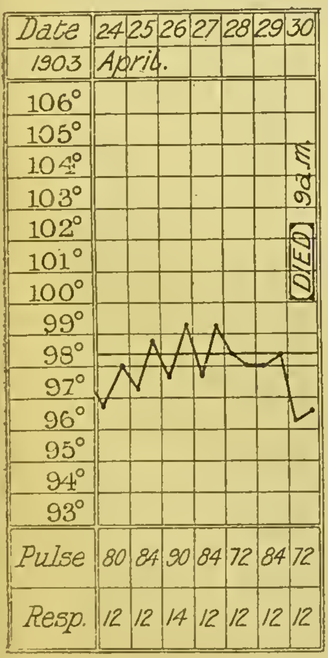
April 30. Died at 9 P.M.

The following chart (p. 61) represents the course of the disease:—

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 29.... | | | | | – |
| Apr. 2.... | | | | | – |
| „ 13.... | | | | | – |
| „ 20.... | + | | + | | + |
| „ 22.... | | – | | | |





May 1, 1903. *Post-mortem*, 9 A.M., 12 hours after death.

The body is that of a well-nourished young man. Rigor mortis is present. There are no bed sores. The pupils are equal and normal. The lymphatic glands are enlarged and the abdomen distended.

*General Condition.*—On opening the body no fluid is found in the peritoneal or pleural cavities. There is a small quantity of clear straw-coloured fluid in the pericardium. The coils of the intestine are much distended, as also is the bladder with urine.

*Circulatory System: Heart.*—Has an excess of fat. There are a few minute petechiæ seen. Otherwise the heart and its structures appear fairly healthy.

*Respiratory System: Lungs.*—Both lungs are congested. There are no signs of pneumonia.

*Liver.*—Weighs 4 lb. Deep dusky red on section, is congested and contains a large quantity of dark blood.

*Spleen.*—Weighs 12 oz. Is enlarged, the capsule being much thickened and adherent. On section the splenic tissue is pigmented and there is an excess of fibrous tissue.

*Kidneys.*—The capsule strips off readily. On section both are highly congested. *Mesenteric Glands.*—Are enlarged.

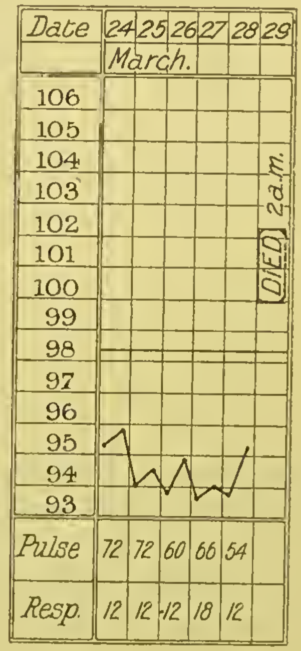
*Brain.*—On removing the dura mater the vessels of the surface of the brain are seen to be intensely injected with blood. The convolutions are flattened and the surface has a dry look. The sulci are mostly obliterated and no excess of sub-arachnoid fluid is seen. On section there is nothing found abnormal in the naked-eye appearances.

*Remarks.*—This was a good typical case of sleeping sickness.

### Case 11.—Goavera (Female). Age 40 years.

March 24. Admitted to hospital. Died on the 29th.

The following chart represents the temperature:—



*Post-mortem* 9 hours after death.

Rigor mortis absent. A small superficial bed sore over left hip. Body extremely emaciated. Enlarged glands (femoral, inguinal, cervical, and posterior occipital).

*Brain.*—Dura mater non-adherent. No change. The vessels are much injected, the sub-arachnoid fluid is greatly in excess, giving opaque glassy look to the surface of the brain, especially over the sulci. Fluid aspirated from the lateral ventricles slightly cloudy.

*Cord.*—Nothing noteworthy.

*Heart.*—Small and flabby. A quantity of deep yellow œdematous jelly-like material at base of heart. Otherwise the heart is normal.

*Lungs.*—Normal.

*Liver.*—Is adherent to the diaphragm, dark in colour, probably due to pigment, but otherwise fairly healthy.

*Spleen.*—Universally adherent and enlarged. On section the splenic substance is slightly pigmented, some excess of fibrous tissue and the pulp rather softer and more friable than normal.

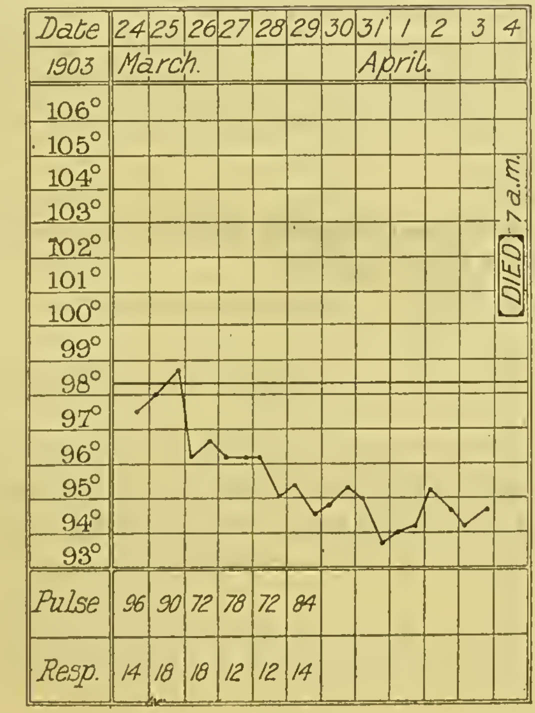
*Kidneys.*—Capsule strips off readily. Substance on section fairly healthy.

*Intestines.*—Large intestine packed with large scybala. Small intestine: many ankylostomata.

Case 17.—Nabujam (Female). Age 30 years. Control. Cerebral Tumour.

March 24, 1903. Admitted to hospital as a case of sleeping sickness.

March 31. Case taken. The patient was suffering from hemiplegia.



Diagnosis.—Cerebral tumour.

The preceding chart (p. 63) represents the course of the disease.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 31.... | + | − | − | | |
| Apr. 1.... | | | | | − |
| " 2.... | | | | | − |
| " 4.... | | | | | − |

April 4. Died at 7 A.M. *Post-mortem*, 9 A.M., 2 hours after death. Rigor mortis absent. Body fairly well nourished.

*Brain.*—On removing the dura mater the small vessels on the surface of the brain were found to be injected. The sub-arachnoid fluid was slightly in excess and appeared dull and opaque. This appearance was not marked as in some former cases, and in fact did not present any very special feature.

There was a large quantity (10 c.c.) of clear fluid in each lateral ventricle. On removing the brain and examining the base, extensive disease was seen in the inter-peduncular region involving the optic chiasma and nerves and floor of the third ventricle. On examining more closely the left hemisphere, this condition was found to be connected with an area of softening and infiltration of the brain substance with a yellow gelatinous gliomatous-like tumour, probably syphilitic in origin.

*Heart.*—Normal.

*Liver.*—Small in size and irregular in shape, due to cicatricial tissue, evidently the site of an old gumma.

*Spleen.*—Normal in size. Adherent to diaphragm. Not pigmented and fairly healthy.

*Kidneys.*—Capsules rather adherent, kidney substance looked normal to the naked eye.

*Urine.*—10 c.c. centrifuged. Red blood corpuscles, white blood corpuscles, epithelial cells, granular casts, but no parasites or ova.

*Remarks.*—This was not a case of sleeping sickness at all, but a tumour of the brain.

### CASE 24.—Yakubu (Male). Age 12 years.

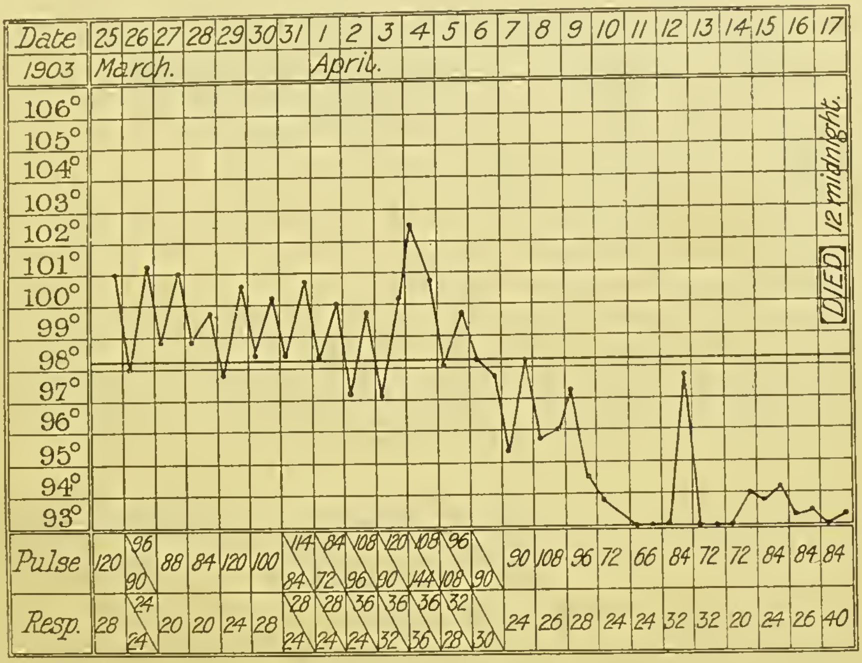
March 25, 1903. Admitted to hospital.

March 27, 1903. 10 c.c. of cerebro-spinal fluid were taken by lumbar puncture, and trypanosomes were found to be numerous in the sediment.

April 7. To-day the patient appears heavy, dull-eyed, and has a discharge from the nose.

April 17. Patient has had convulsions for several days. He died at 12 P.M.

The following chart represents the course of the disease:—



The following table shows the presence or absence of trypanosoma in the cerebro-spinal fluid and in the blood:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903.<br>Mar. 23.... | | | | | + |

April 18. *Post-mortem* 9 A.M.

The body is well nourished. Rigor mortis is present. There are no bed sores. The left pupil is a little larger than the right.

*Heart.*—There is an increase of fat about the base of the heart. There is no jelly-like deposit and no petechiæ in the endocardium. The heart substance and valves are normal.

*Lungs.*—There is no fluid in either plural cavity.

Right. Lymphatic glands at the root of the lung are enlarged, otherwise this lung is healthy.

Left. Deep congestion of the posterior part of the upper lobe, and the whole of the lower lobe.

*Spleen.*—Is enlarged, and on section is pigmented and tough.

*Liver.*—Somewhat enlarged, congested, and with fatty degeneration.

*Kidneys.*—Both healthy.

*Intestines.*—There is no fluid in the peritoneal cavity. The mesentery especially between the transverse colon and the duodenum is spotted over with white patches varying from a pin's head to a pea. On section there is no feeling of cutting through calcareous deposit.

The mesenteric glands are enlarged to the size of a large bean. (Ankylostomata (6 or 7) were found in the jejunum.)

*Brain.*—The surface is injected and the sub-arachnoid fluid is in excess, giving the sulci a cloudy ground-glass appearance. On section the naked-eye appearances are normal.

*Remarks.*—There is nothing much to be remarked in this case. The temperature chart shows the usual fall before death. Trypanosomes were found in the cerebro-spinal fluid before death, and a streptococcus was obtained from the heart's blood and lateral ventricles after death.

### CASE 25.—Kimbra (Male). Age 30. Occupation, Wood-cutter. District, Bussi. Food, Fish, Bananas, and Sweet Potatoes.

March 25, 1903. Admitted to hospital.

April 6. Patient has a weak tottering gait, is heavy and dull-eyed. His tongue is tremulous. His speech is a far-away, high-pitched, indistinct monotone. His lips are dry and cracked, and he passes urine involuntarily.

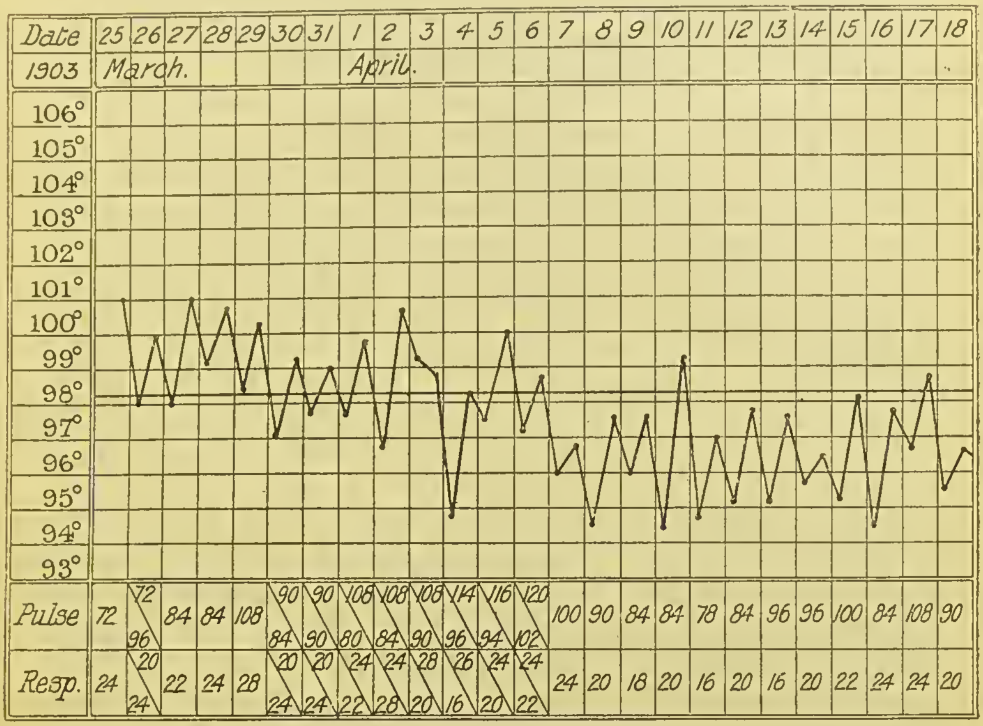
He states he has been ill 7 months, and that a brother died of sleeping sickness in the same house.

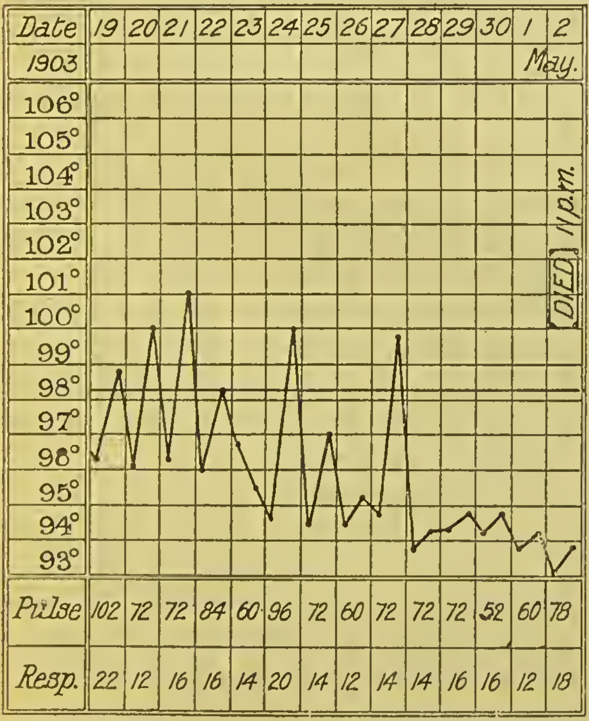
April 22. *General Condition.*—He is well nourished. His gait is weak and tottering, and he can hardly walk without assistance. He is fairly intelligent. The expression of his face is heavy, sad, and dull. There is no œdema of any part. The colour of the mucous membrane is normal. There are no trophic changes. The lymphatic glands are generally enlarged.

*Nervous System.*—His intelligence and memory are fair. He sleeps much during the day. His eyes are normal. He has marked tremor of his tongue, but none of lips or hands. Knee-jerks are present; no ankle clonus.

*Alimentary System.*—His appetite is good, and his tongue is moist and furred. The liver and spleen are not enlarged. He requires medicine every two or three days to move his bowels.

*Circulatory System.*—Heart sounds weak, but normal, apex beat





imperceptible. Pulse 90, tension fair. Size medium, compressible, and regular.

*Respiratory System.*—Normal.

*Cutaneous System.*—His skin is normal and there are no eruptions.

The preceding chart (p. 67) shows the course of the disease.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 26.... | | | | − | + |
| Apr. 20.... | + | | + | | |
| ,, 22.... | | − | | | |

May 3. *Post-mortem* 11 hours after death.

The body is that of a well-nourished, powerful, muscular young man. Rigor mortis is absent. There are no bed sores and the skin is smooth and healthy. The pupils are equal and normal. The superficial lymphatics are enlarged (the size of beans).

On opening the body, no fluid is found in the peritoneal cavity, nor in either pleural cavity. A small quantity of clear straw-coloured serum is in the pericardium.

*Left Lung.*—Weighs 12 oz., and is normal.

*Right Lung.*—Weighs 16 oz., and is slightly congested, otherwise normal.

*Heart.*—There is excess of fat about the base of the heart. The muscular substance is somewhat pale, but healthy, and the valves are normal. There are some petechiæ on the pericardium on the outside of the left ventricle, but none on the endocardium. Weight 9 oz.

*Spleen.*—Weighs 8 oz. Is universally adherent, and the capsule is thickened and contracted in places, giving the organ a lobed appearance. On section there is an excess of fibrous tissue, and some pigmentation.

*Liver.*—Weighs 3 lb. 6 oz. The capsule is somewhat thickened and is adherent to some of the underlying structures.

On section there is advanced fatty degeneration.

*Kidneys.*—Each weighs 4 oz. The capsules strip off easily, the organ is healthy.

*Brain.*—The vessels on the surface are deeply injected. The sub-arachnoid fluid in excess. The convolutions are flattened. There is a great excess of fluid in the lateral ventricles, otherwise there is nothing abnormal to the naked eye.

*Alimentary System.*—The mesenteric glands are enlarged. The whole of the large intestine is packed full of hard scybala.

*Remarks.*—This is an ordinary case of sleeping sickness.

### CASE 22.—Zebuganza (Male). Age 40 years. District, Entebbe. Food, Sweet Potatoes and Bananas.

March 25, 1903. Admitted to hospital.

April 7. His gait is weak, he has a heavy look, and his speech is indistinct and weak. There are no marked tremors. He states that he has been ill 2 months.

April 22. *General Condition.*—Patient is poorly nourished. There is slight dementia. His gait is weak and staggering, and the expression on his face is vacant. There is no œdema anywhere. The mucous membrane is normal, and there are no trophic changes. Supra-condylar, inguinal, submental and the glands in the posterior triangle of the neck are enlarged. He complains of pain in the back and head.

*Nervous System.*—His intelligence and memory are impaired. He lies about and sleeps all day. His speech is indistinct and mumbling. His eyes are normal. The tongue is moist and tremulous. There are tremors of the lips and hands and general muscular tremors. The knee-jerk is present, ankle clonus absent.

*Alimentary System.*—His appetite is poor. Stomatitis is present. The liver and spleen are not enlarged.

*Circulatory System.*—Heart sounds are normal, but feebly heard owing to emphysema, there is no bruit. The pulse is 88, feeble, no tension, irregular in force and regular in rhythm.

*Respiratory System.*—Emphysema in both lungs.

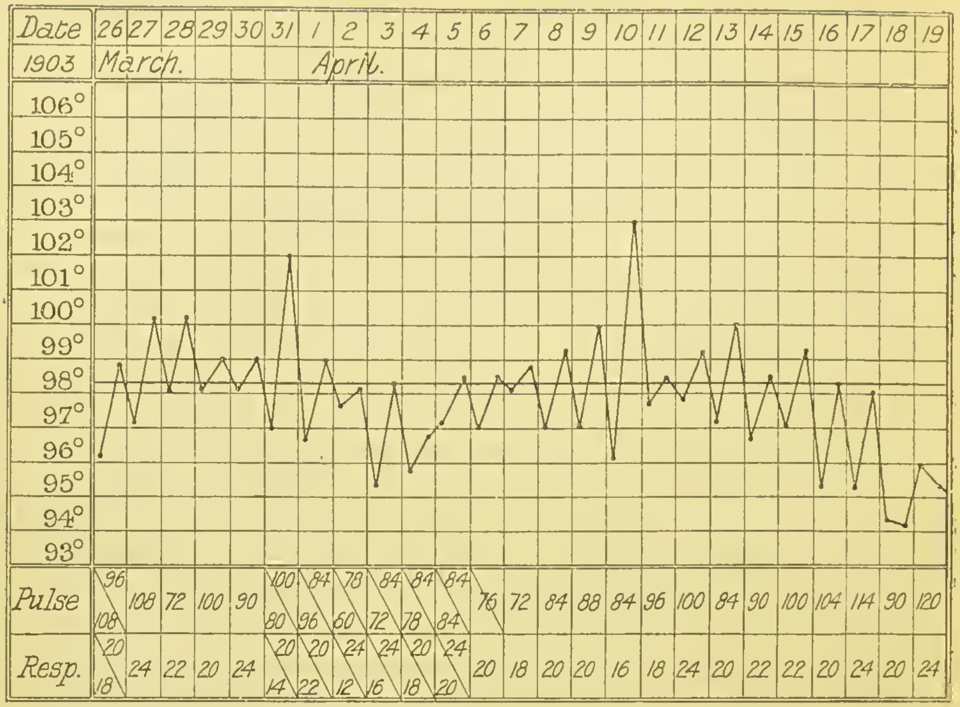
*Skin.*—Is dry and slightly scaly.

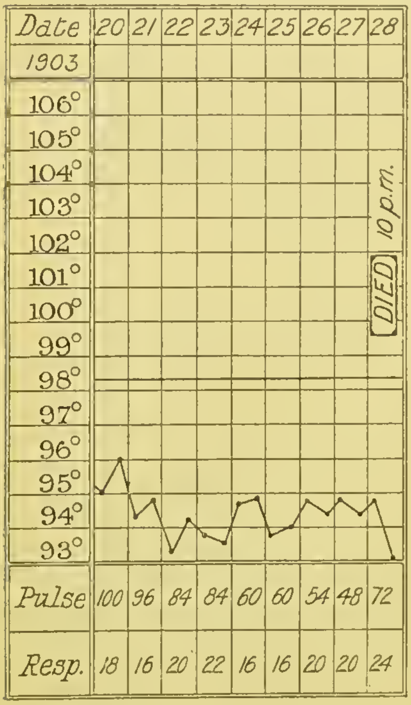
The following chart (p. 70) represents the course of the disease.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 27.... | | | | – | + |
| Apr. 18.... | | | + | | |
| ,, 22 ... | | – | + | | |
| ,, 29*... | + | – | – | | |

* Blood taken 15 hours after death.





April 29. *Post-mortem* 11½ hours after death.

The body is that of an elderly man, apparently 40 or 50 years of age. Rigor mortis is present. There are no bed sores. The abdominal region is retracted, the body is thin, but not emaciated. The pupils are equal and normal. The inner aspect of the lips is ulcerated. The superficial lymphatic glands are slightly enlarged.

On making an incision from the chin to the pubes, no fluid is found in the peritoneal cavity. The spleen is somewhat enlarged, the bladder is distended with urine, the mesenteric glands are enlarged to the size of a bean, and the descending colon contains some hard fæces. On removing the sternum, both lungs are found to be adherent to the thorax by old tough adhesions. There is no fluid in either pleural cavity. There is half-an-ounce of clear straw-coloured serum in the pericardium.

*Heart.*—Is normal in size, flabby in consistence, and there is a slight increase of fat at the base. The muscular substance of the wall of the left ventricle appears fairly healthy. The aortic and mitral valves are normal. The right ventricle contains a large pale-coloured clot. The pulmonary and tricuspid valves are healthy. There are no signs of petechiæ at any part of the endocardium. A slight thickening is seen at the attachment of the valves of the aorta. The heart weighs 8 oz.

*Lungs.*—Left lung weighs 8 oz. There is some emphysema along the anterior border of the apex. On section the lung tissue is fairly healthy. The lymphatic glands at the root of the lung are enlarged and pigmented.

Right lung weighs 16½ oz. There is emphysema along the anterior margin and at the apex, the lower lobe is deeply congested. Large quantities of frothy fluid exude on pressure. The upper lobe is also congested, but to a less extent.

*Spleen.*—Weighs 6 oz. It is slightly enlarged. On section the substance is slightly pigmented and is fairly normal in consistence.

*Kidneys.*—Left weighs 4 oz. The capsule strips off readily. On section there is slight congestion, otherwise healthy.

Right, weighs 2½ oz., condition same as left.

*Liver.*—Weighs 2 lb. 12 oz. The capsule is smooth and somewhat dark in colour. On section the substance of the liver is found to be slightly pigmented, and there is fatty degeneration.

*Brain.*—On removing the calvarium the dura mater is found to be healthy. The vessels of the surface are injected and the convolutions are flattened. The sub-arachnoid fluid is in excess, distending the sulci and giving them the opaque ground-glass appearance. On section the substance appears fairly healthy. There is a quantity of fluid in the lateral ventricles. The brain is firm in consistence, and nothing further of importance was found.

*Remarks.*—This is an ordinary uncomplicated case of sleeping sickness, which probably lasted 3 months.

Case 41.—Warasansa (Male). Age 32. District, Bussi. Occupation, Bark-cloth Maker. Food, principally Sweet Potatoes.

March 25, 1903. Admitted to hospital. He states that none of his family have died of sleeping sickness, and no other cases have occurred in his house. He thinks the illness began about three months ago, the first symptoms being headache and a tendency to sleep.

April 6. His gait is undecided and weak, pulse 132.

April 28. *General Condition.*—He is fairly well nourished, he is up and about, but his gait is weak and staggering. His intelligence seems good. The expression of his face is wistful. There is slight general enlargement of the lymphatic glands.

*Nervous System.*—His intelligence is good, and memory fair. He sleeps a good deal during the day. His speech is rather weak and monotonous, but distinct. He has a jerky tremor of the tongue, but not of lips or hands. The knee reflexes are present.

*Alimentary System.*—Tongue moist, covered with white fur. His liver is normal, but his spleen is palpable.

*Circulatory System.*—The impulse of the heart is neither visible nor palpable. Heart sounds are normal. No bruit. Pulse is 80 per minute, size fair, tension moderate, regular.

*Cutaneous System.*—Scaly eruption on upper part of chest.

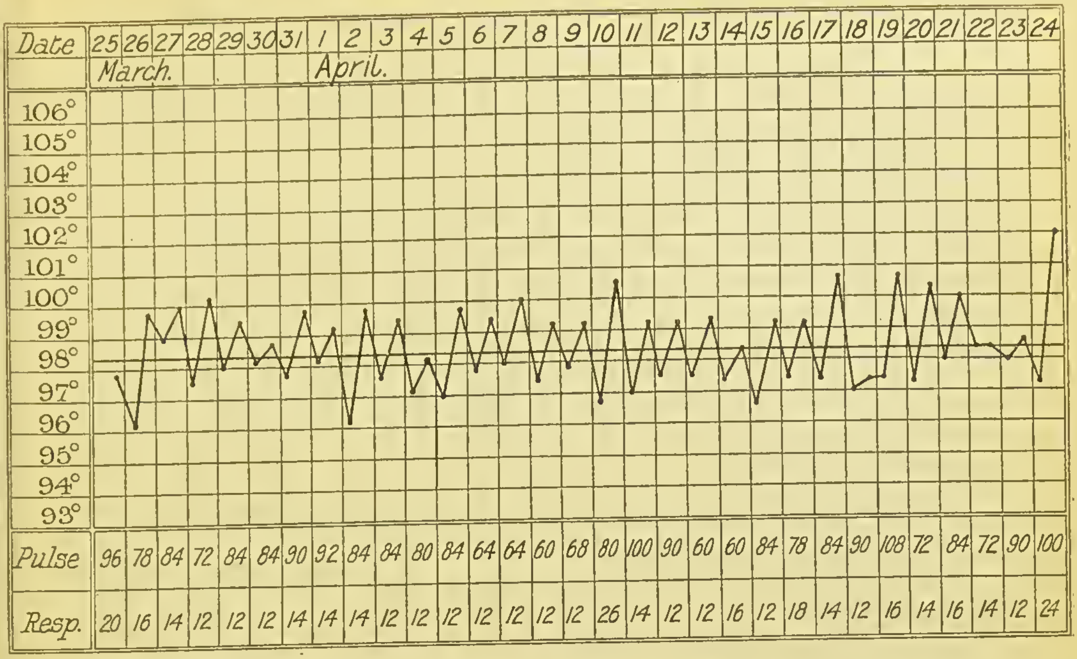
May 13. During the past week patient has become considerably worse. He passes his motions under him. His temperature is almost constantly below 96° F. On the 11th inst. 50 c.c. of fluid was obtained by lumbar puncture. This morning there is a staring vacant expression of the face and the upper eyelids are retracted. Patient is unable to stand. There is a painful swelling of the lymphatic glands of the right femoral and inguinal region. There is no fluctuation. Nothing external can be seen to account for this inflammation. There is no chancre, nor wound of any kind of leg or foot. After death these inflamed glands were found to be breaking down into pus.

May 14. Died at 9 A.M. No *post-mortem.*

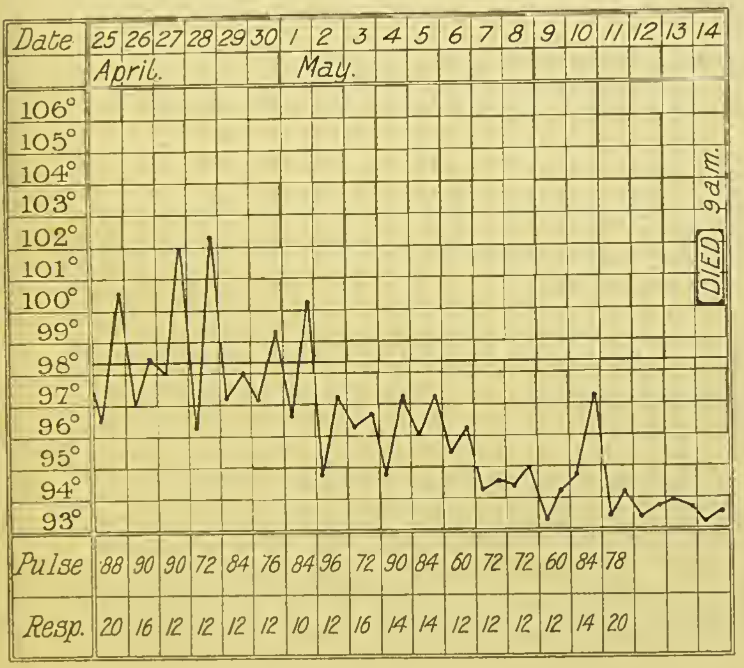
The following chart (p. 73) represents the course of the disease.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 6.... | | | | | – |
| " 12.... | + | | – | | |
| " 19.... | | – | | | |
| " 22.... | + | – | + | | |
| May 11.... | | | | | + |



| Date | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  | March. |  |  |  |  |  |  | April. |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Pulse | 96 | 78 | 84 | 72 | 84 | 84 | 90 | 92 | 84 | 84 | 80 | 84 | 64 | 64 | 60 | 68 | 80 | 100 | 90 | 60 | 60 | 84 | 78 | 84 | 90 | 108 | 72 | 84 | 72 | 90 | 100 |
| Resp. | 20 | 16 | 14 | 12 | 12 | 12 | 14 | 14 | 14 | 12 | 12 | 12 | 12 | 12 | 12 | 12 | 26 | 14 | 12 | 12 | 16 | 12 | 18 | 14 | 12 | 16 | 14 | 16 | 14 | 12 | 24 |



| Date | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  | April. |  |  |  |  |  | May. |  |  |  |  |  |  |  |  |  |  |  |  |  |
| Pulse | 88 | 90 | 90 | 72 | 84 | 76 | 84 | 96 | 72 | 90 | 84 | 60 | 72 | 72 | 60 | 84 | 78 |  |  |  |
| Resp. | 20 | 16 | 12 | 12 | 12 | 12 | 10 | 12 | 16 | 14 | 14 | 12 | 12 | 12 | 12 | 14 | 20 |  |  |  |

*Remarks.*—This case resembles Case 35 (Fatoma), in the acute inflammation of lymphatic glands which at death are found to be suppurating. Whether this is due to the disease or to some coincident cause it is difficult to say. Except for this it is a simple uncomplicated case of sleeping sickness.

Case 37.—Kiagabidoia (Male). Age 50 years. District, Bussi. Occupation, Fisherman. Food, Fish, Bananas, and Sweet Potatoes.

March 25, 1903. Admitted to hospital. He states he has been ill more than two months. There had not been any other cases of sleeping sickness in the same house.

April 15. *General Condition.*—Development and nutrition poor. Gait unsteady and weak, but not very typical. Intelligence fair. Expression of face dull, but not characteristic of sleeping sickness. No œdema. Glands generally enlarged. Complains of pain in thighs, but no headache. Pulse 74. No cutaneous eruptions.

*Nervous System.*—Intelligence and memory fair. Sleep not excessive. Speech normal. Eyes normal. Tongue jerky. Knee-jerk present. Ankle clonus absent.

*Alimentary System.*—Appetite good. Tongue moist and furred. Liver and spleen not enlarged.

*Circulatory System.*—Impulse of heart cannot be felt. Sounds normal.

*Skin.*—Scaly on legs.

May 4. Tongue still tremulous. Marked tremor of hands. His speech is weak and monotonous. He is reported as not sleeping during the day.

May 6. He is becoming very feeble and tremulous.

May 20. There is right facial paralysis to-day. He cannot walk now.

May 22. Died at 11 p.m.

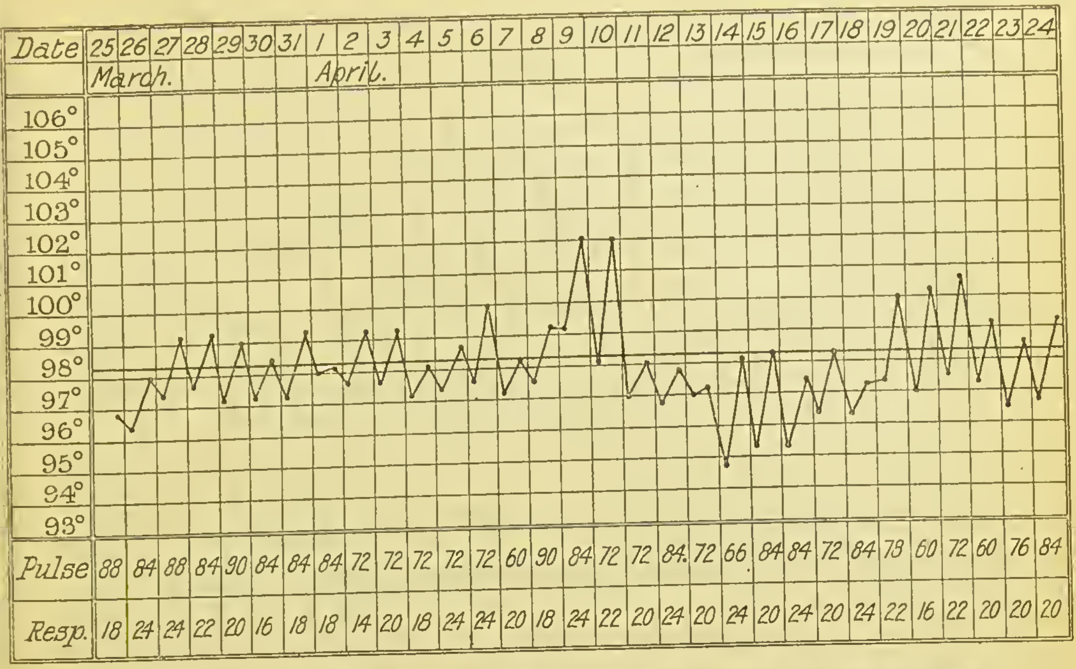
The following chart (p. 75) represents the course of the disease.

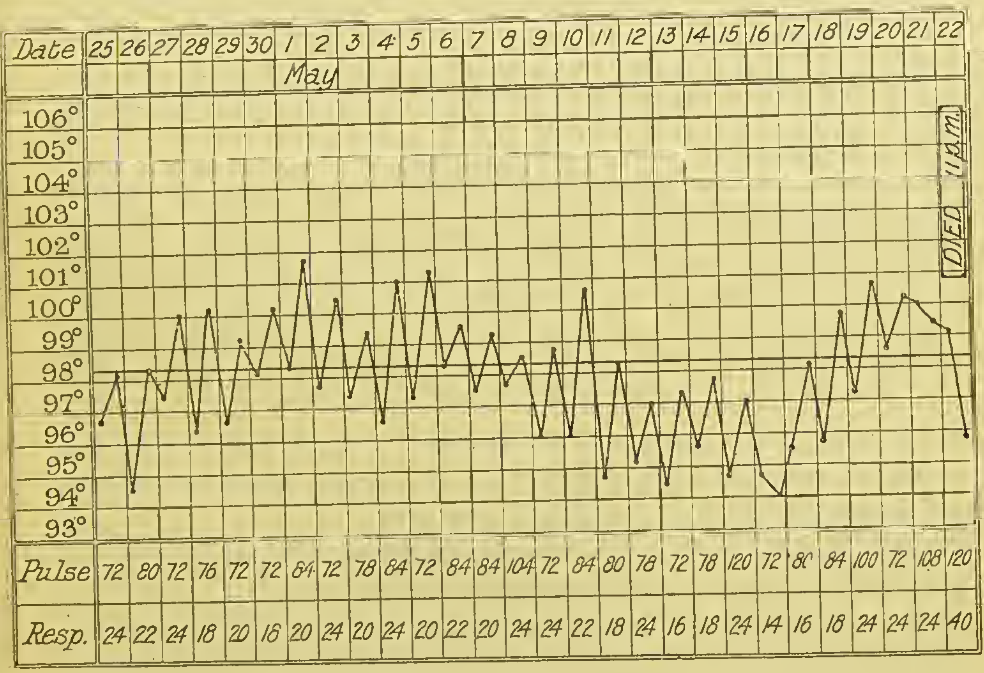
The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 28.... | | | | | – |
| Apr. 1.... | | | | | – |
| ,, 2.... | | | | | – |
| ,, 13.... | | | | | – |
| ,, 22.... | | – | | | |
| May 4.... | | | | | + |

May 23. *Post-mortem* 12 hours after death.

Rigor mortis present. The body is that of an oldish man, poorly nourished. Chest rigid and barrel shaped. Abdomen retracted. The





right eye is closed owing to swelling of upper eyelid. Superficial glands enlarged.

On opening the body in the usual way, the lungs overlapped the heart to a large extent. No free fluid in pleural or peritoneal cavities, and about ½ oz. in pericardium.

*Heart.*—Weight 8 oz., the right ventricle is soft and flabby and shows signs of atrophy of its substance, which in places is converted into a brownish-yellow gelatinous tissue. The coronary arteries and branches are rigid and tortuous. Interior of heart looks normal. No petechiæ. Substance dark and firm.

*Lungs.*—Right weighs 12 oz., left 10 oz. Both are emphysematous, and there is marked congestion of the lower lobe of each lung.

*Liver.*—The surface is roughened, like early stage of a "hobnailed" liver. On section the substance is firm and feels uneven to the touch. Weight 3 lb.

*Spleen.*—Capsule smooth. Substance breaks down readily. Markedly pigmented. Weight 8 oz.

*Kidneys.*—Each weighs 3½ oz. Congested. Capsule strips readily.

*Pancreas.*—Rather yellow in colour, otherwise healthy.

*Mesenteric Glands.*—Enlarged. Mesenteric and intestinal vessels congested and tortuous.

*Brain.*—On removing the dura mater the surface of the brain is seen to be covered with greenish pus (in the sub-arachnoid spaces). The convolutions are flattened and the sulci obliterated. On the upper surface of the cerebellum and the adjacent under surface of the cerebrum the sub-arachnoid pus is most abundant. On removing the brain from the body, a considerable quantity (? 2 oz.) of turbid fluid escaped from the brain and spinal canal. Cerebral substance softened. Lateral ventricle, aqueduct of Sylvius and fourth ventricle all considerably dilated. Vessels of circle of Willis rigid and patent.

*Remarks.*—For some time it was doubtful if this was a case of sleeping sickness, especially as it was only after repeated examination that the trypanosomes were found in the cerebro-spinal fluid. This case, like some of the others, ended in purulent meningitis.

### CASE 38.—Kitaroma (Male). Age 20 years. District, Bussi. Occupation, Fisherman.

March 25, 1903. Admitted to hospital. States that no friends had died of sleeping sickness, and that the illness began six months before admission, the first symptom being fever.

*General Condition.*—He is fairly well nourished. His gait weak and uncertain, intelligence fair, expression of face dull.

There is no œdema. The mucous membranes are normal in colour. No trophic changes. Lymphatic glands generally enlarged, to the size of beans in the groin. He complains of pain in the occipital region and back of the neck.

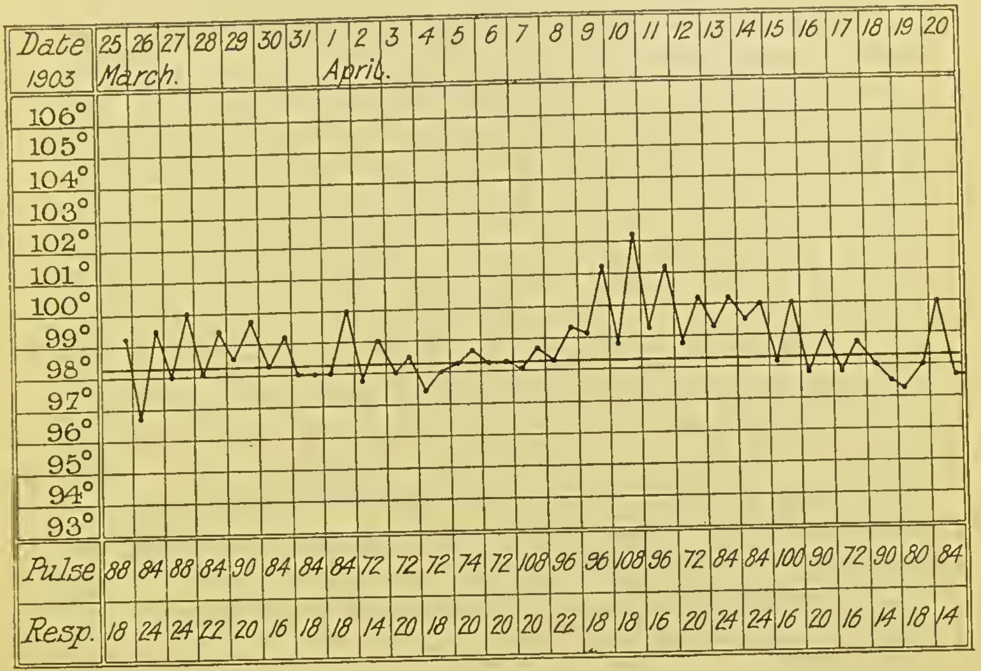
*Nervous System.*—His intelligence is fair, but he sleeps too much during the day. His speech is weak, indistinct and monotonous. Eyes normal. There is marked tremor of the tongue and some of the hands. Knee-jerk is present, ankle clonus absent.

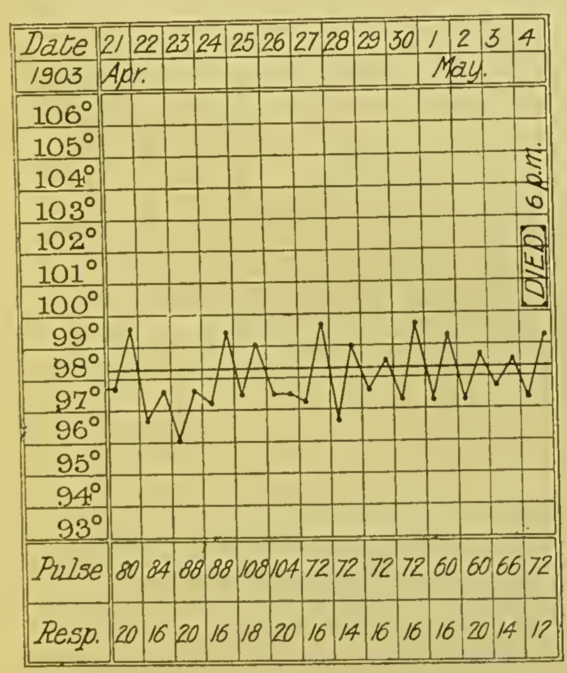
*Alimentary System.*—His appetite is good, tongue moist and furred. Liver and spleen are enlarged.

*Circulatory System.*—Heart sounds very weak, apex beat almost imperceptible and no bruit. Pulse 84 per minute lying down, weak, regular, low tension, easily compressible.

*Skin.*—Rather dry and scaly.

The following chart represents the course of the disease:—





The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 10 .... | + | | − | | |
| " 13 .... | | | | | + |
| " 20 .... | + | − | − | | |
| " 22 .... | | − | | | |
| " 27 .... | + | − | + | | |

May 5. *Post-mortem* 20 hours after death.

A fairly well nourished young man about 20 years of age. Rigor mortis present. No bed sores. Superficial lymphatic glands enlarged.

On opening the body there is no fluid found in either the peritoneal or pleural cavities. There is ½ oz. of straw-coloured fluid in the pericardium.

*Heart.*—There is an excess of fat at the base and some deposit of black pigment under the pericardium. No petechiæ, outside or in. The muscular substance appears healthy, valves normal. Weight 10 oz.

*Lungs.*—Right weighs 15 oz. Some congestion of the upper lobe. The posterior part of the inferior lobe is deeply congested, although not actually consolidated. Left weighs 15 oz. Lower lobe deeply congested, upper lobe also, but to a less extent.

*Spleen.*—Weighs 12 oz. Capsule smooth. On section the tissue is tough, and there is an excess of fibrous tissue.

*Liver.*—Weight 3 lb. 3 oz. Adherent to the diaphragm about an inch from the anterior border. The falciform ligament was œdematous. The organ contains a large quantity of blood, it is highly congested, and probably there is some fatty degeneration.

*Kidneys.*—Each weighs 3½ oz. They are intensely congested, and deep dusky red in colour.

*Mesenteric Glands.*—Are somewhat enlarged (size of beans).

*Brain.*—The vessels are injected on the surface, and the convolutions are somewhat flattened, otherwise the appearances are fairly normal. On section the brain appears normal to the naked eye.

*Remarks.*—The accompanying photographs were taken on May 4. (See Plates 6 and 7.)

CASE 15.—Waiswa (Male). Age 10 years. District, Busoga (Wakelai).

March 25, 1903. Admitted to hospital. States that his illness began with headache about February 20.

April 7. He has a sad heavy look and cracked lips. Pulse 132.

April 18. *General Condition.*—He is well nourished and walks fairly well. His intelligence is fair, but he has a dull expression. There is no œdema. The lymphatic glands generally are enlarged. The pulse is 100 and fair. There are no cutaneous eruptions.

*Nervous System.*—He is always drowsy. The speech is not altered. The tongue is tremulous. Knee-jerk is present, ankle clonus absent.

*Alimentary System.*—His appetite is good, bowels regular, and no enlargement of liver or spleen.

*Circulatory System.*—Much exaggerated pulmonary second sound. Pulse a little irregular, tension fair, size fair.

*Skin.*—Normal.

May 6. Disease is in the late first stage.

May 13. During the past week the patient has rapidly got worse and now is unable to stand, evidently passing into the third stage. He sleeps practically all day.

May 16. This morning patient is unconscious and does not respond or appear to pay attention when spoken to. His head is drawn down to the right shoulder, and there are jerky twitchings of the arms.

May 18. Died at 10 P.M.

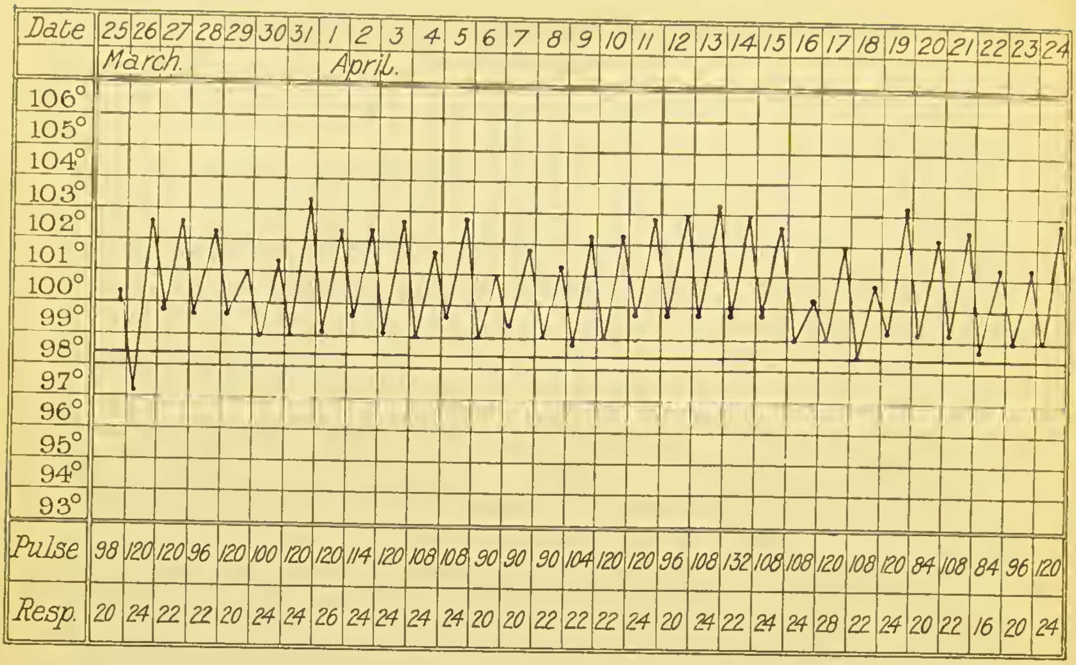
The following chart (p. 80) represents the course of the disease.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

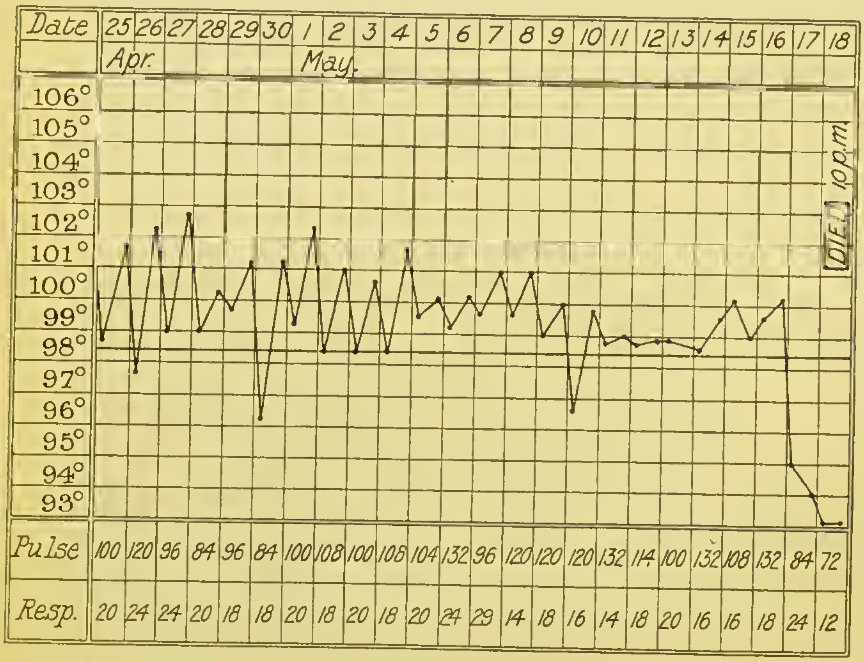
| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 29.... | | | | | + |
| Apr. 18.... | + | | + | | |
| ,, 22.... | | – | | | |
| May 14.... | | | | | + |

May 19. *Post-mortem* 12 hours after death.

Rigor mortis is absent. The body is poorly nourished and there is marked retraction of the abdomen (as there was during life for the last few days). The superficial glands are only slightly enlarged. There are no bed sores.



| Date | 25 March | 26 | 27 | 28 | 29 | 30 | 31 | 1 April | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 98 | 120 | 120 | 96 | 120 | 100 | 120 | 120 | 114 | 120 | 108 | 108 | 90 | 90 | 90 | 104 | 120 | 120 | 96 | 108 | 132 | 108 | 108 | 120 | 108 | 120 | 84 | 108 | 84 | 96 | 120 |
| Resp. | 20 | 24 | 22 | 22 | 20 | 24 | 24 | 26 | 24 | 24 | 24 | 24 | 20 | 20 | 22 | 22 | 22 | 24 | 20 | 24 | 22 | 24 | 24 | 28 | 22 | 24 | 20 | 22 | 16 | 20 | 24 |



| Date | 25 Apr. | 26 | 27 | 28 | 29 | 30 | 1 May. | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pulse | 100 | 120 | 96 | 84 | 96 | 84 | 100 | 108 | 100 | 108 | 104 | 132 | 96 | 120 | 120 | 120 | 132 | 114 | 100 | 132 | 108 | 132 | 84 | 72 |
| Resp. | 20 | 24 | 24 | 20 | 18 | 18 | 20 | 18 | 20 | 18 | 20 | 24 | 29 | 14 | 18 | 16 | 14 | 18 | 20 | 16 | 16 | 18 | 24 | 12 |

The body was opened in the usual way. The peritoneal cavity contains no free fluid, nor is there any fluid in the pericardial or pleural cavities.

*Heart.*—Weighs 5½ oz. There is a small sub-pericardial ecchymosis on the left ventricle near the base. No petechiæ. The valves are healthy, and the muscle substance normal.

*Lungs.*—Each weighs 5 oz. Normal.

*Liver.*—Is smooth on the surface. On section it is slightly pigmented. The substance is very tough. Weight 11 lb. 14 oz.

*Spleen.*—Weighs 6½ oz. Is not adherent. Just about the middle of the external surface, under the capsule is a small hard tumour the size of a pea. Near the hilum is a similar hard tumour the size of a filbert nut. On section the tumours are red in colour, divided into loculi by trabeculæ of connective tissue, and are circumscribed, not invading the splenic pulp. Substance of spleen is tough and not obviously pigmented.

*Kidneys.*—Each weighs 2 oz. Normal.

*Pancreas.*—Is firm and deep yellow in colour.

*Intestines.*—Looked normal. *Mesenteric Glands.*—Are enlarged and some have hæmorrhages into them.

*Brain.*—On removing the dura mater the surface of the brain is seen to be deeply congested all over. There is slight excess of subarachnoid fluid, with ground-glass opacity, in the sulci. The convolutions are a little flattened. Nothing abnormal was found in the interior of the brain, except intense congestion of the choroid plexuses.

The ventricles did not contain any excess of fluid.

*Remarks.*—This is a typical case of sleeping sickness.

The accompanying photograph was taken on May 4, 1903. (See Plate 8.)

### Case 45.—Leobeni (Male). Age 25 years. District, Bussi.

March 26, 1903. Admitted to hospital. No information can be got from this man, as he cannot speak.

April 1. *General Condition.*—This man is well nourished, although so advanced in the disease that he can neither walk nor stand. The expression of his face is dull and vacant, and he fixes you with an expressionless stare. There is no œdema of any part. The colour of the mucous membrane is normal. There are no trophic changes. The lymphatic glands in the anterior triangle, the inguinal and femoral regions are slightly enlarged. There are no cutaneous eruptions, and the skin itself appears normal.

*Nervous System.*—It is difficult to say what amount of intelligence this man retains, as it is impossible to get him to answer questions. When asked anything he looks like a man just awakened from a sound sleep: he looks stupid and dazed, his eyes wander from face to face in a helpless, staring, wondering way. In regard to sleep, he is reported to sleep well at night, but not much during the day. His powers of speech have entirely disappeared, but when spoken to sharply he still is able to murmur indistinctly his name. His eyes are normal. There is extreme tremor of tongue, lips, hands, and generally all over his body. He is still sensible to touch, and his muscle sense is fairly normal. Both knee-jerks are increased, the right more than the left, and ankle clonus is present in both ankles, also in the right more than the left.

*Alimentary System.*—He is still able to drink milk. His tongue is moist, furred, and extremely tremulous. Liver and spleen are not enlarged.

*Circulatory System.*—Heart. The apex beat can neither be seen nor felt. On auscultation the sounds of the heart are distant and weak, pulse 160, tension low, small in size, easily compressible and regular.

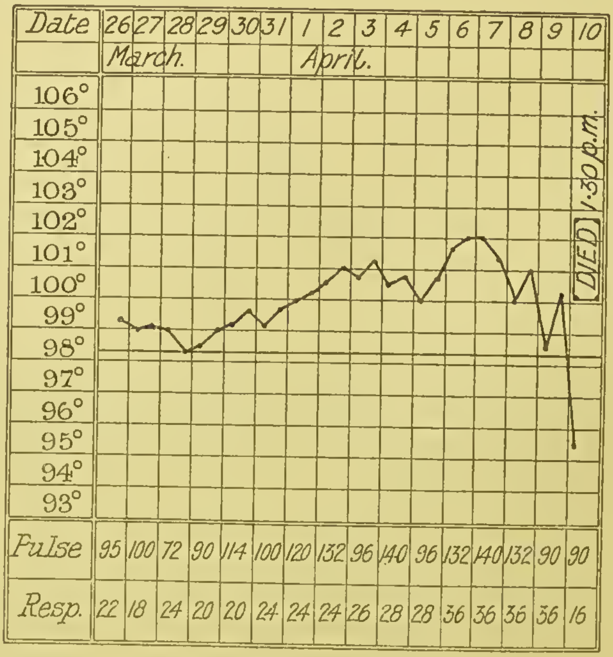
*Respiratory System.*—Nothing noteworthy.

*Skin.*—The skin appears normal, moist, and smooth.

*Ophthalmoscopic Examination.*—Left eye, slight blurring of disc. Right eye could not be seen.

April 10. Died 1.30 P.M.

The following chart represents the course of the disease :—



The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 28.... | | | | | + |
| Apr. 1.... | | | | – | |

April 10. *Post-mortem* immediately after death :—

The body is well nourished; there is a bed sore beginning on the right hip.

*Brain.*—On removing the calvarium the dura mater presents nothing noteworthy; but on removing that membrane the surface of the brain is found to be covered with a layer of greenish-coloured pus. This matter is contained in the sub-arachnoid spaces and is in excess of the normal sub-arachnoid fluid. The vessels on the surface of the brain are injected. On removing the brain the base is found to be in the same condition of purulent meningitis. The fluid in the ventricles is found to be cloudy. The substance of the brain is somewhat softened.

*Cord.*—The sub-arachnoid or cerebro-spinal fluid in the vertebral canal in the lumbar region was distinctly purulent.

*Heart.*—External fat, yellow in colour, is in excess. Muscular substance appears healthy. No petechiæ on inner surface. Valves healthy.

*Lungs.*—Right, healthy. Left, apex of lower lobe is consolidated, airless, and a greenish-yellow fluid is expressed on pressure. A piece floats in water. This patch of pneumonia is as large as a small orange.

*Liver.*—Large, dark in colour, gall bladder distended. On section dark, pigmented, and congested.

*Spleen.*—Enlarged, pigmented, congested, and friable.

*Kidneys.*—Capsule strips readily. Congested, otherwise healthy.

*Remarks.*—Noteworthy on account of the invasion of pneumococcus which probably gave rise to the meningitis.

This brain, like Kaperi's, is not at all typical (see Plate 9). We have not sent a drawing of an ordinary Sleeping Sickness brain, but shall do so.

A photograph of one is given on Plate 10.

### Case 36.—Dikodemo. Male. Age 25 years.

Occupation, Teacher. Protestant School. District, Nkumba. Food, Fish, Bananas, and Sweet Potatoes.

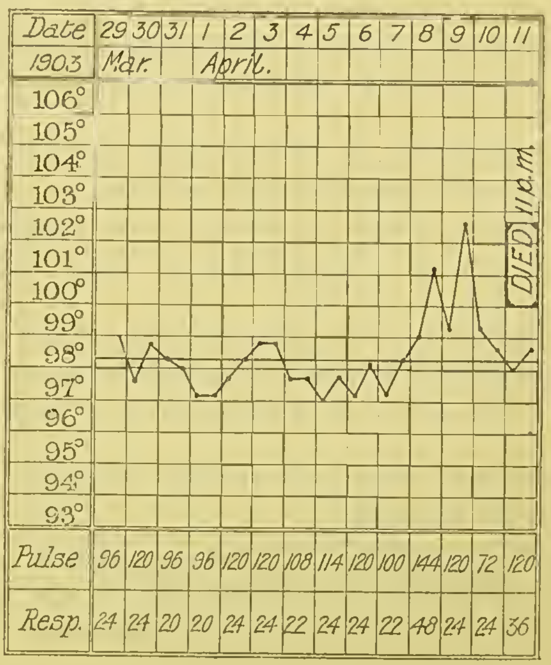
March 29, 1903. Admitted to hospital. Disease, sleeping sickness. States duration of illness about two months.

*Present Condition.*—Dikodemo is a man of good development and is well nourished. He stands with difficulty with feet apart and knees bent, balancing himself. When he walks his gait is weak, staggering, and uncertain. When asked to shut his eyes, his feet being together, he retains his balance. His facial expression is dazed, dull, wistful, and he stares vacantly now at one, now at another, with his eyes wide open and unblinking. It is difficult to get an answer from him.

There is no œdema or puffiness of any part. His mucous membrane is normal in colour. There are no trophic changes. The lymphatic glands in the occipital region, angle of jaw, and axilla are not enlarged, but in the anterior triangle of the neck and groin they are felt to be slightly enlarged, about the size of a small bean.

April 11. Died at 11 P.M.

The following chart represents the course of the disease :—



The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 3.... | + | − | − | − | + |
| „ 7.... | | | − | | |
| „ 12.... | | | | | − P.M. |

April 12. *Post-mortem* 12 hours after death.

The body is that of a well-nourished man of middle height and apparently about 25 years of age. Rigor mortis is present. There are no bed sores.

*Brain.*—On removing the calvarium the dura mater appears healthy. The surface of the brain has an excess of fluid, the vessels are injected, especially on the left side. The sub-arachnoid fluid is fairly clear, but in some of the sulci it has an opaque ground-glass appearance. The lateral ventricles contain an excess of fluid of a slightly cloudy character. The substance of the brain is not softened, and nothing further abnormal was noted.

*Heart.*—Healthy.

*Spleen.*—Slightly enlarged and tougher than normal.

*Liver.*—Pigmented. *Kidneys.*—Healthy.

*Lungs.*—There is some congestion at the base of the left lung. Purulent fluid extrudes from the small bronchi on pressure.

*Remarks.*—This is a typical case of sleeping sickness. It is to be noted that the trypanosoma is not found in the cerebro-spinal fluid taken twelve hours after death.

The trypanosomes seem to disappear from the body fluids soon after death.

### CASE 42.—Katola (Male). Age 25 years. District, Bussi. Occupation, Charcoal Burner. Food, Sweet Potatoes.

March 25, 1903. Admitted to hospital. He states that none of his family have died of sleeping sickness, nor have any other cases occurred in his house. He also states that he was taken ill 3 months before admission to hospital, and that his first symptom was fever.

April 6. He is thin. He walks fairly. Speaks well, but has tremor of tongue and hands. Pulse 140.

April 29. *General Condition.*—This man is badly nourished, his ribs are all showing. He is not confined to bed, and walks fairly well. His intelligence is fair, but the expression of his face is somewhat dull and heavy. His lymphatic glands are enlarged to the size of pigeon's eggs in the femoral region, and that of beans at the angle of the jaw.

*Nervous System.*—He is fairly intelligent and his memory is normal. He is reported not to sleep excessively during the day. His speech is weak, but distinct. There are tremors of the tongue and hands. The knee-jerks are present, and ankle clonus absent.

*Alimentary System.*—His appetite is good. His tongue moist and furred. His liver is not enlarged, but his spleen is palpable.

*Circulatory System.*—The impulse of the heart is seen and felt in the fifth space. There is an apical systolic murmur, and also a localised presystolic murmur near the ensiform cartilage. Pulse 112, size fair, regular.

May 6. During the last few days this patient has become much more feeble and can hardly walk a few paces without clinging to something for support. Tremor of tongue and fingers very marked.

May 13. No change in condition.

May 15. About 50 c.c. of cerebro-spinal fluid obtained by lumbar puncture. Specific gravity 1002.

May 20. Patient is passing into the third stage.

May 28. Died at 11 P.M.

The following chart (p. 87) represents the course of the disease.

The following table shows the presence or absence of trypanosomes :—

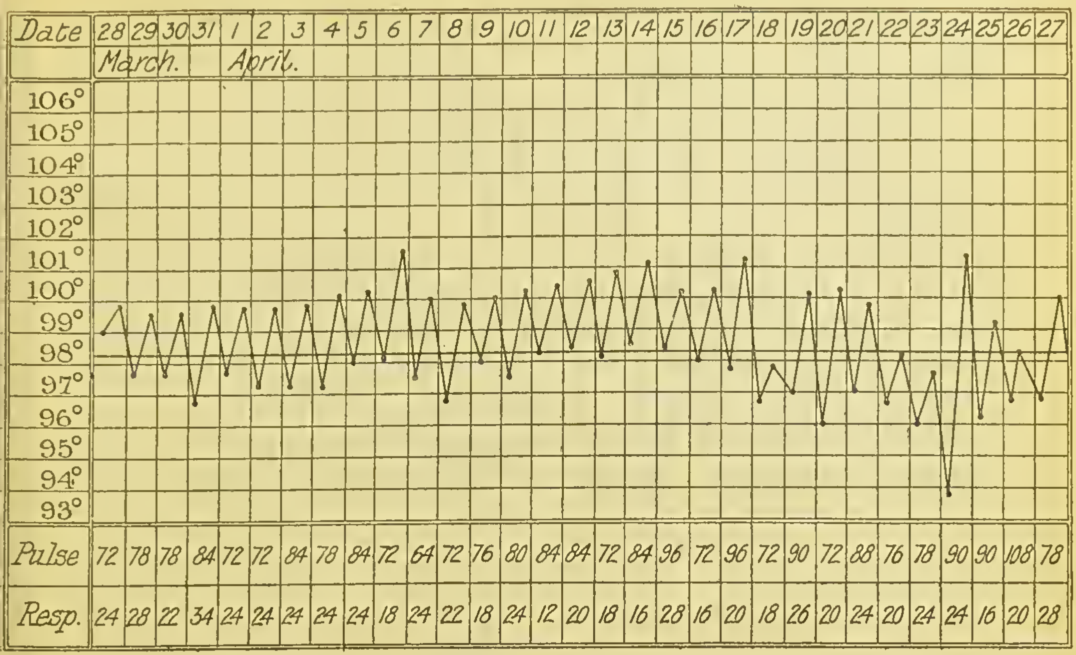
| Date. | Parasites in blood. | | | Parasites in C. S. fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 6 . . . . | | | | | + |
| „ 19 . . . . | | – | | | |
| „ 22 . . . . | + | – | + | | |
| May 15 . . . . | | | | | + |

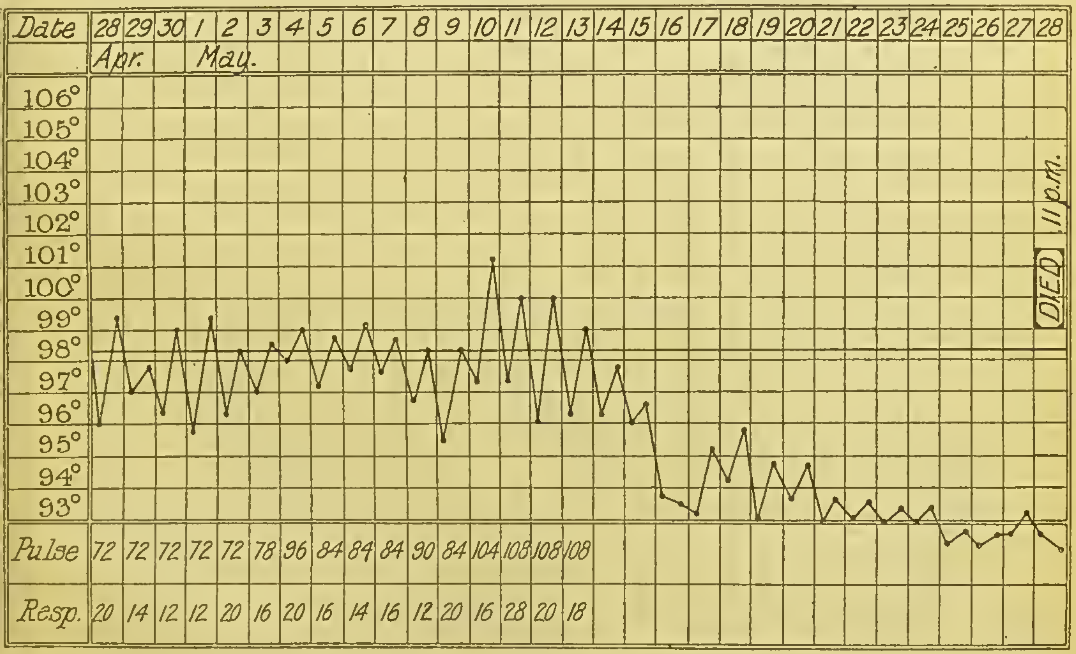
May 29. *Post-mortem*, 10 A.M., 11 hours after death.

The body is that of a fairly well nourished man. There are no cutaneous eruptions, and no bed sores. Rigor mortis passing off. Slight enlargement of superficial lymphatic glands.

On opening the body in the usual way, no fluid was found in the pleural, pericardial, or peritoneal cavities.

*Heart.*—Weighs 8 oz. There are no petechiæ on the surface of the interior, no disease of orifices or valves, and the muscular substance looks healthy.





*Lungs.*—Left. Adherent to parietes, especially towards the base. Weighs 10 oz. Base somewhat congested. Glands markedly enlarged.

Right. Adherent at apex and towards base. Weighs 10 oz. Both lobes are healthy.

*Liver.*—Adherent to diaphragm on the right side, old perihepatitis. The left lobe is adherent to the spleen. Weighs 3 lb. 8 oz. It is somewhat congested, otherwise normal. Not abnormally tough or fibrous.

*Spleen.*—Generally adherent to diaphragm, also to liver. Considerably enlarged. Weighs 1½ lb. On section looks dark and breaks down readily under the finger.

*Kidneys.*—Each weighs 4 oz. Capsules strip off readily. Healthy on section. *Pancreas.*—Firm, yellow in colour.

*Intestines.*—Look normal externally. *Mesenteric Glands.*—Slightly enlarged.

*Brain.*—Dura mater not adherent. Convolutions flattened. Marked injection of vessels of pia mater. No marked increase of sub-arachnoid fluid. The lateral ventricles are dilated. Fourth ventricle not dilated. Brain otherwise normal, except for some hæmorrhages in the caudate nucleus of both hemispheres. *Vide* photograph, Plate 10.

*Remarks.*—10 c.c. of cerebro-spinal fluid was taken 11 hours after death and centrifuged. No living trypanosomes were found.

*Bruce & Nabarro* *Sleeping Sickness. Pl. I.*

*see p. 25.*

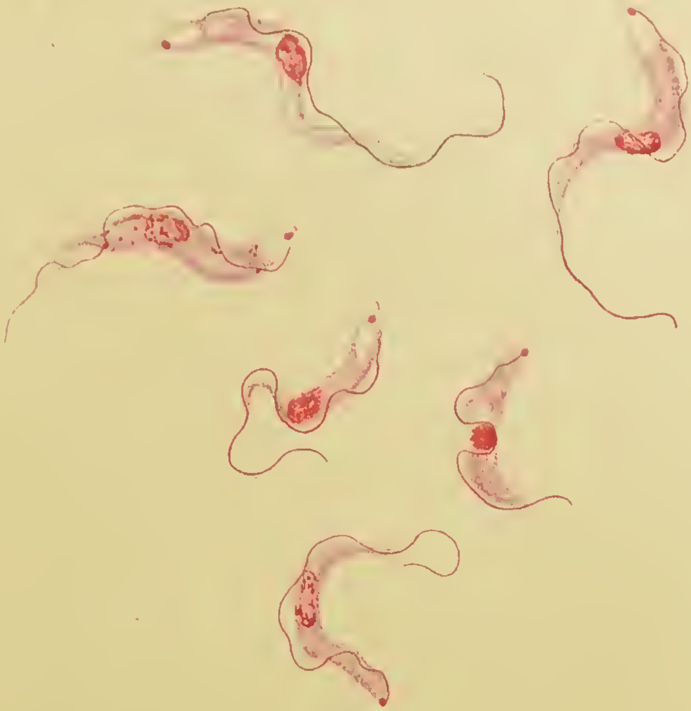


Fig. 1. × 2000

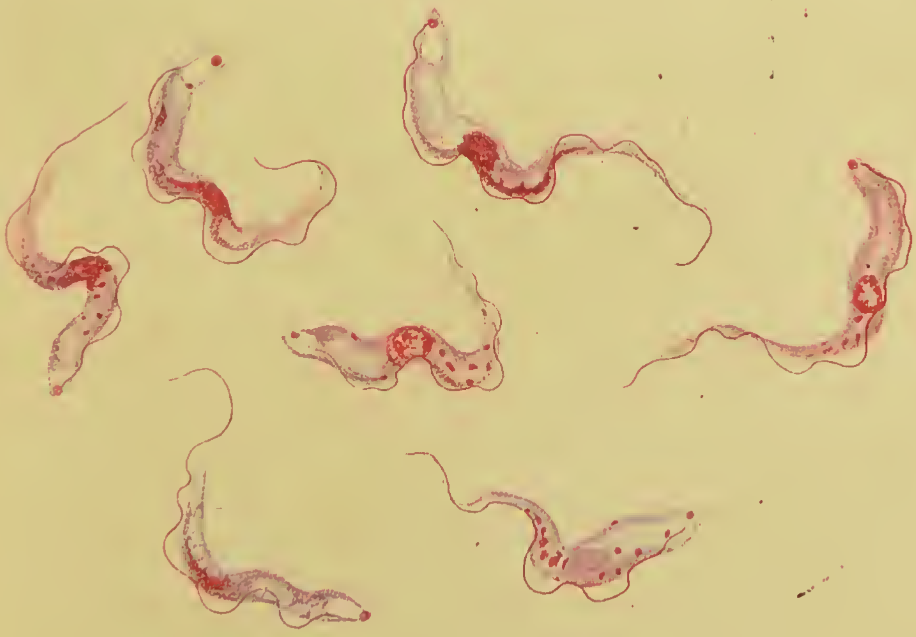


Fig 2. × 2000

Huth, lith. et imp.

*Bruce & Nabarro* *Sleeping Sickness Pl. 2*

*(see p 25)*

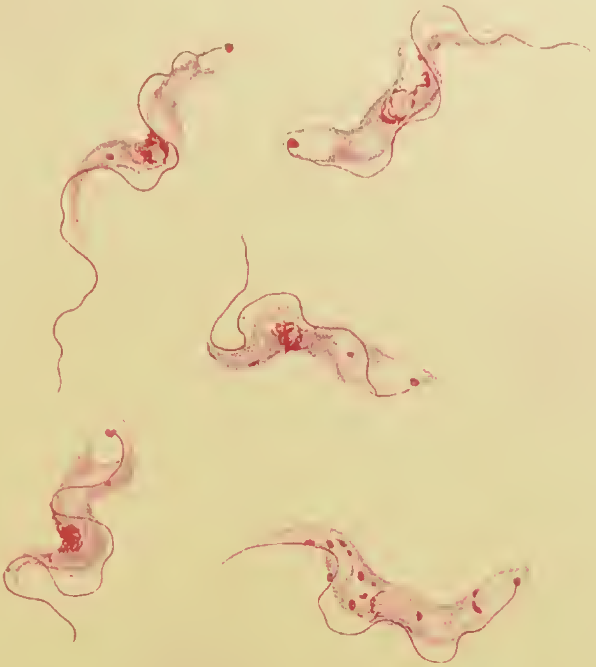


Fig. 3. × 2000

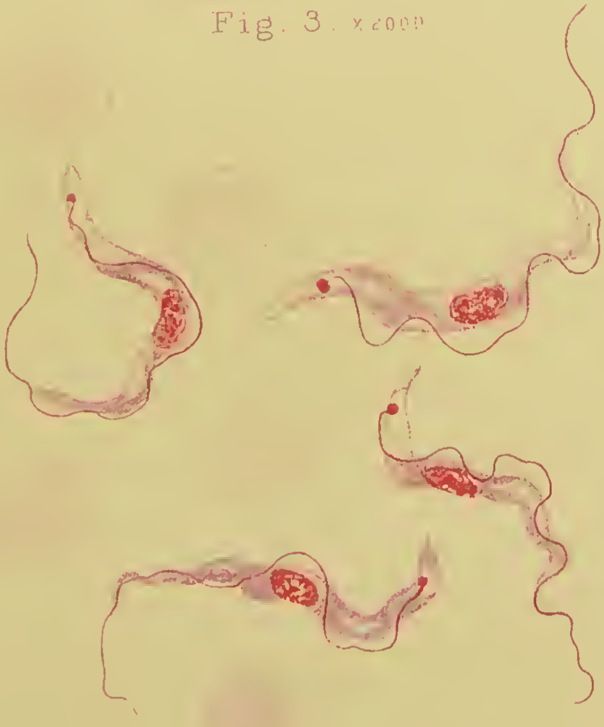


Fig. 4. × 2000

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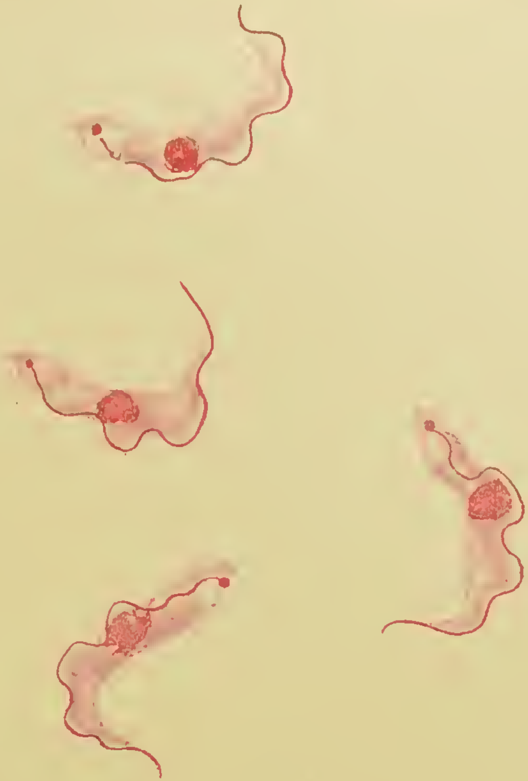

Fig. 5. ×2000.

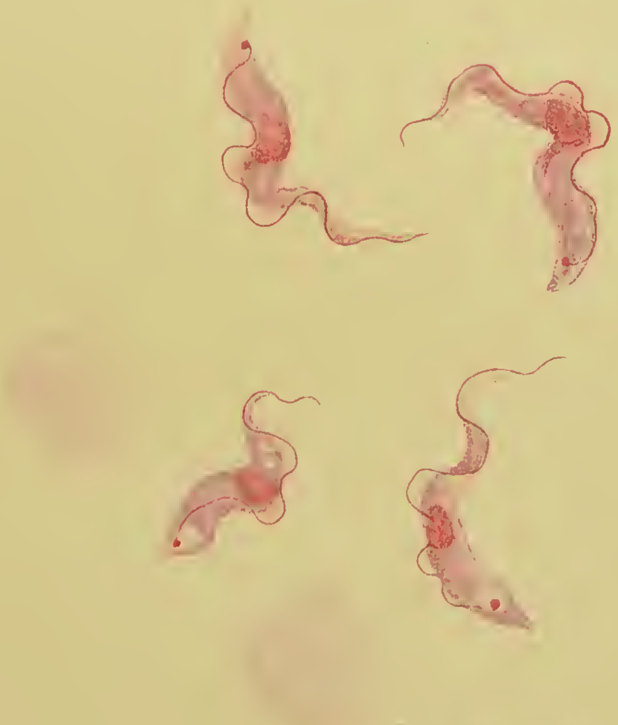

Fig. 6. ×2000.

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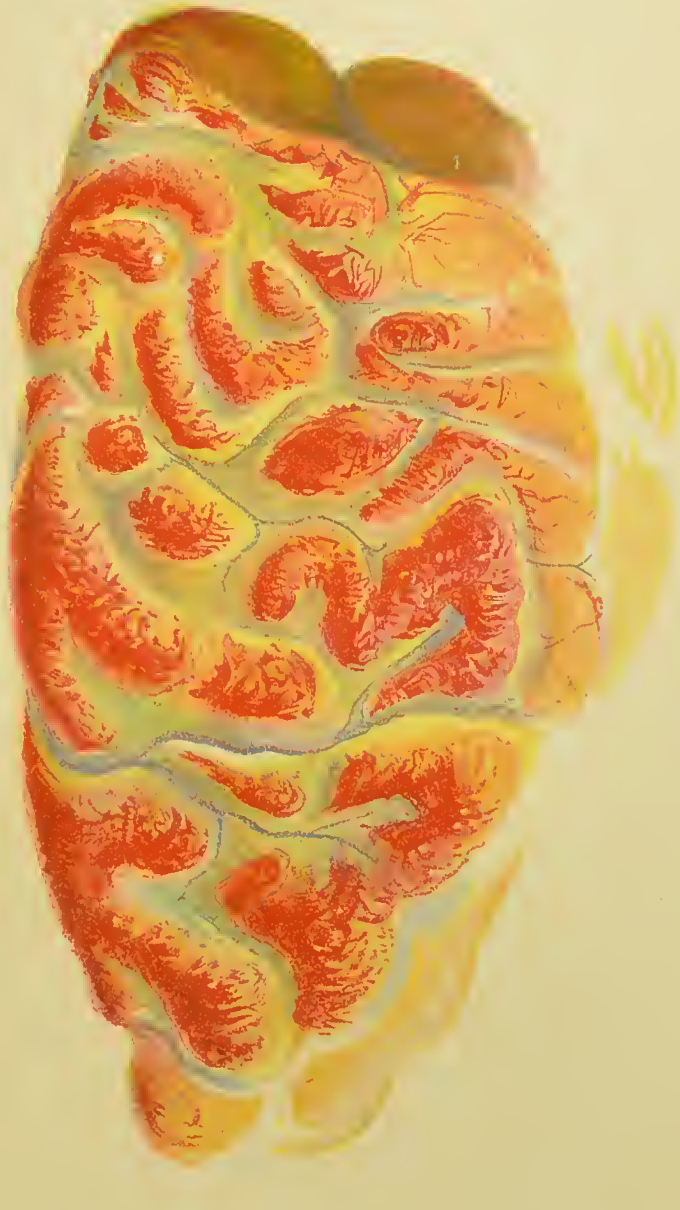
Figs. 7 and 8.—MONKEYS USED IN EXPERIMENTS.

[illegible] Sleeping sickness Pl. [illegible]
(see p. [illegible]



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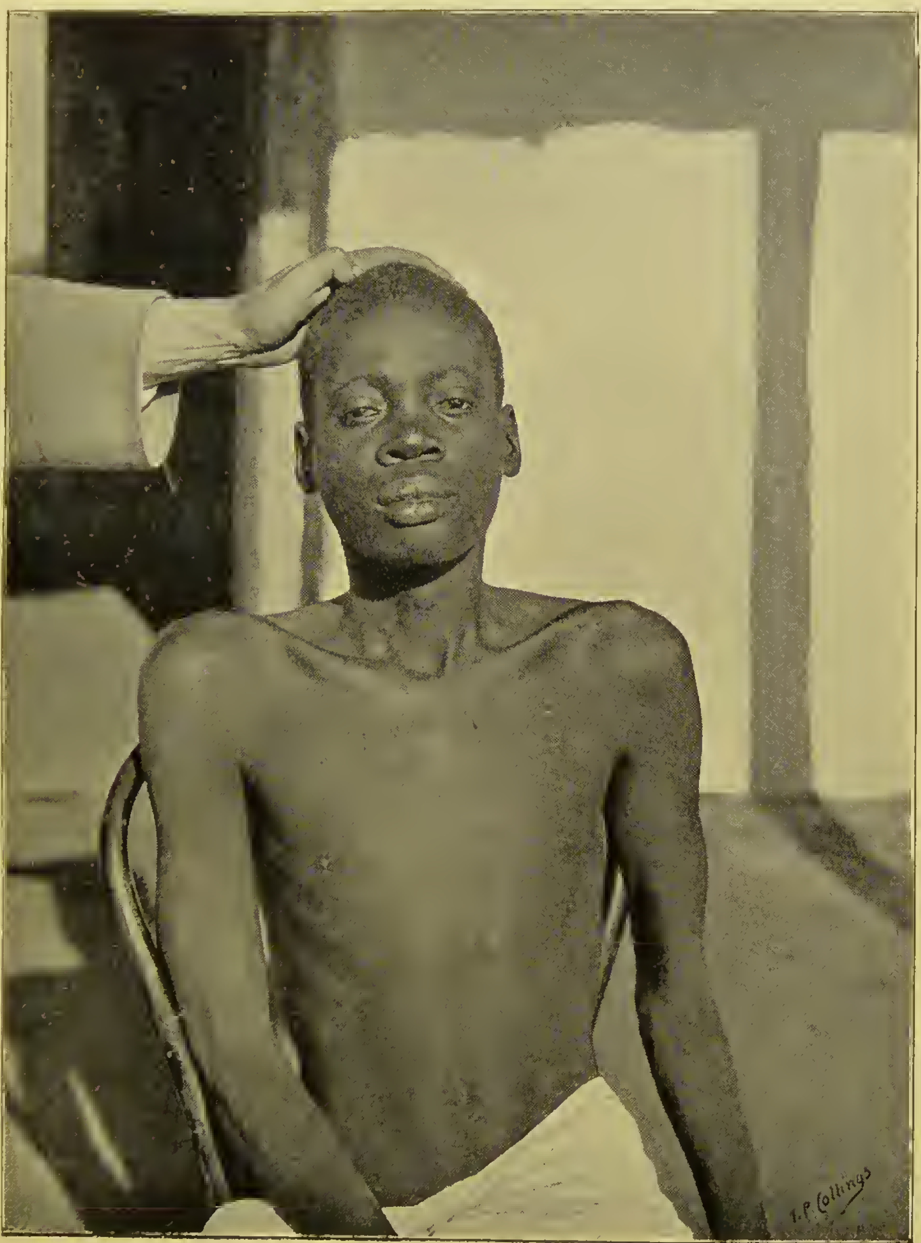
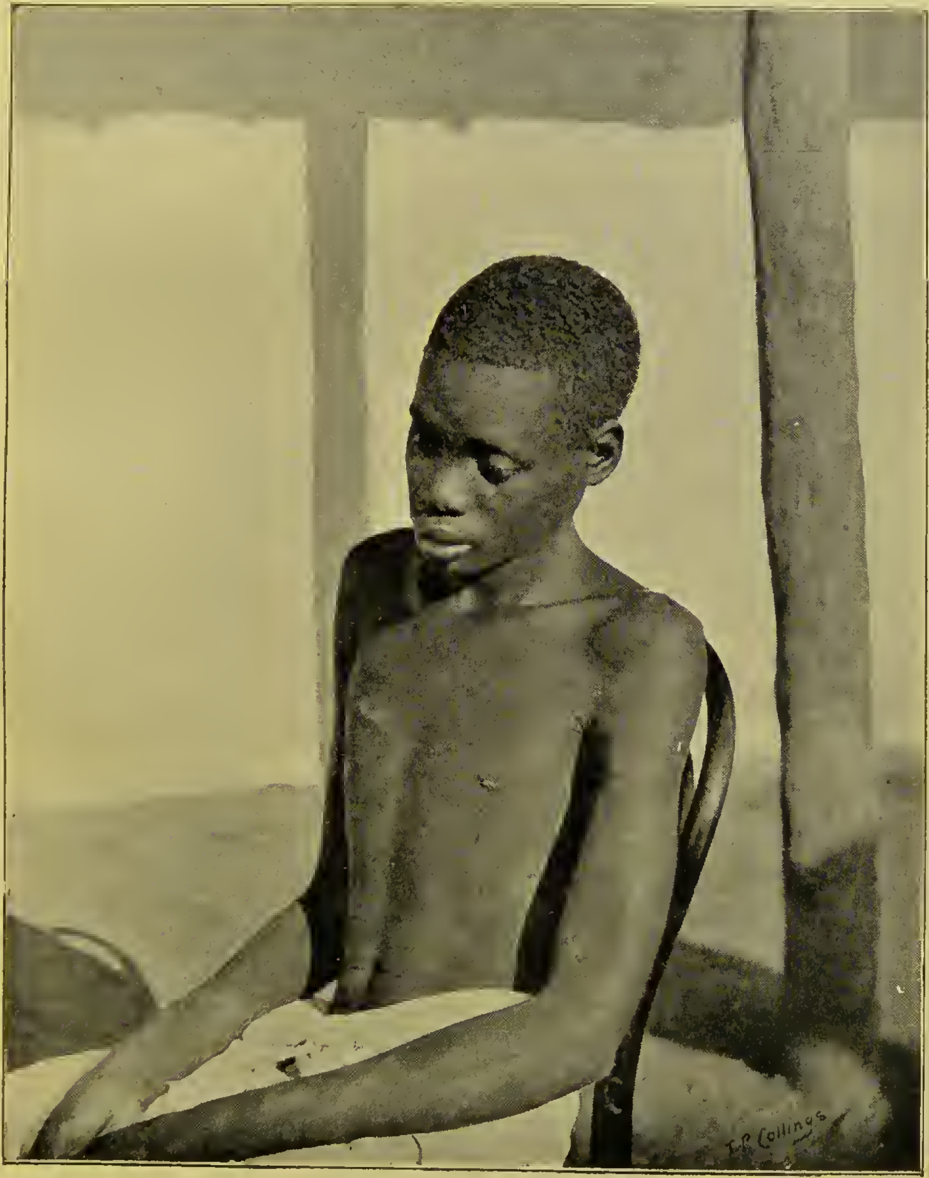
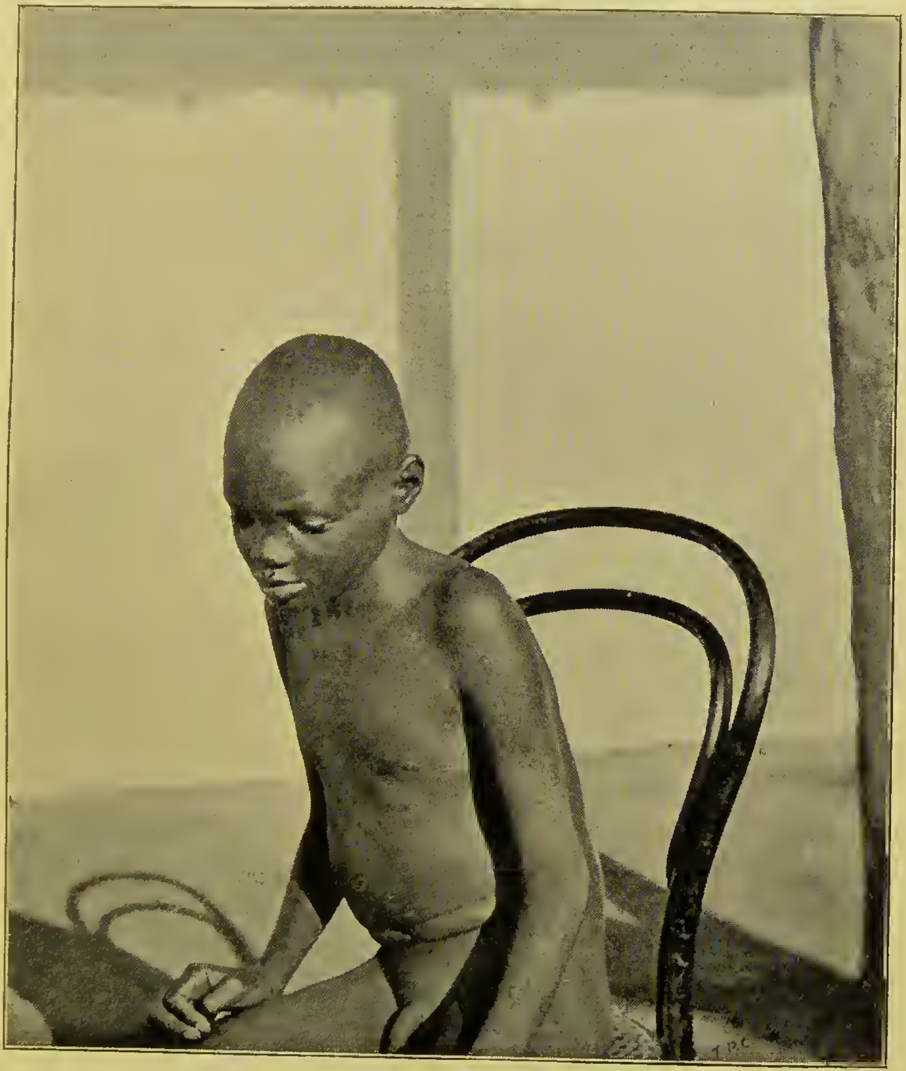
SURFACE OF BRAIN
CASE 4, PAPER



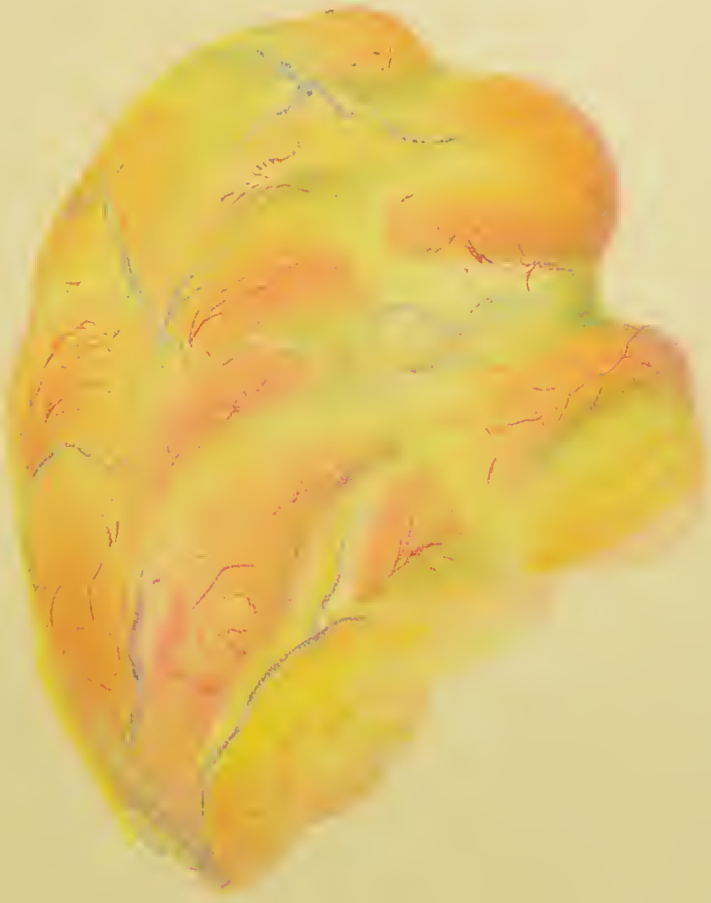
CASE 38. KITAROMA.



Case 38. Kitaroma.



CASE 15. WAISWA.



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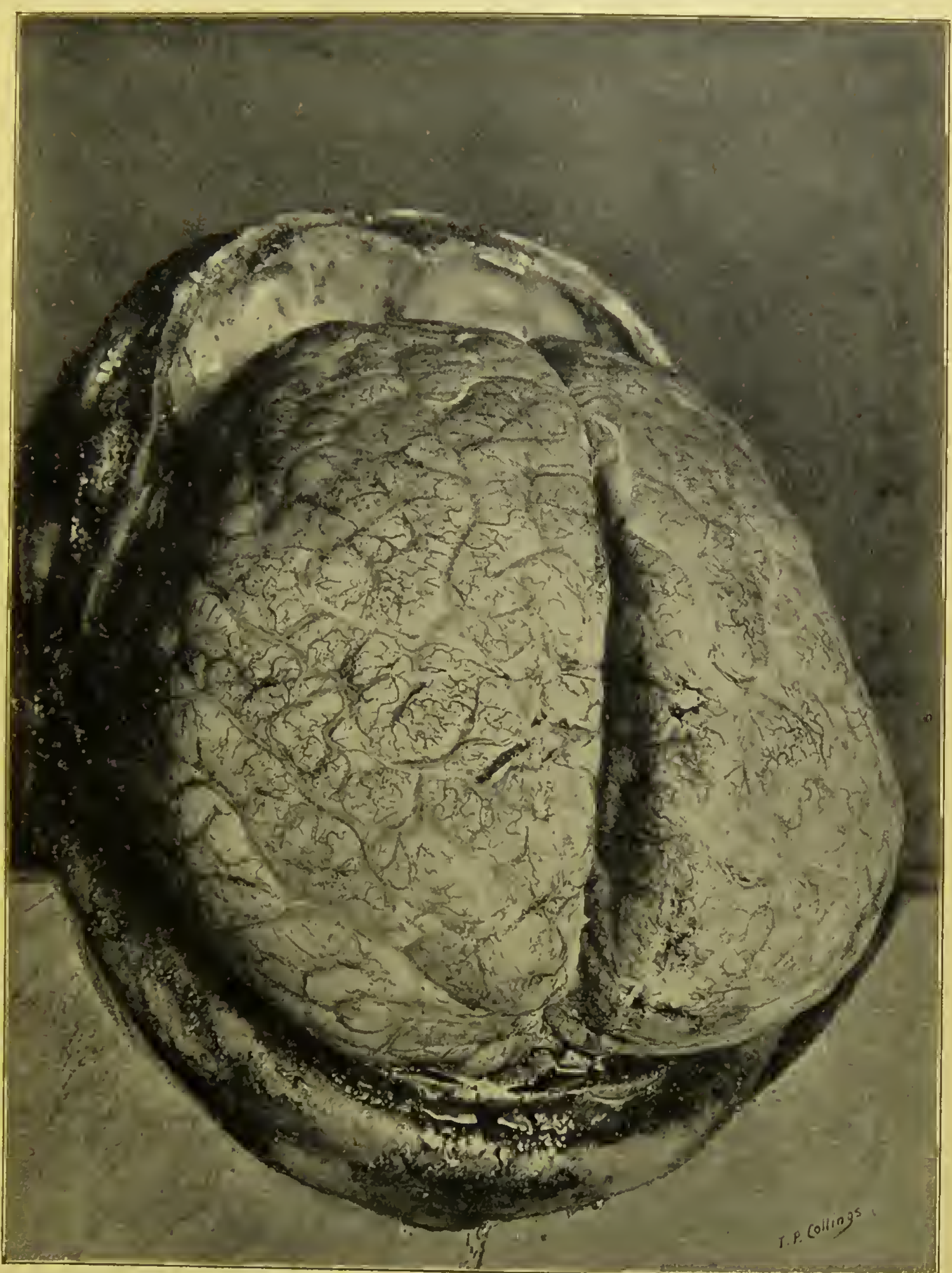
SURFACE OF BRAIN
CASE 85 ITTOBEIL.



PHOTOGRAPH OF BRAIN. CASE 42. KATOLA. SHOWING FLATTENING OF CONVOLUTIONS AND INJECTION OF VESSELS.

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2. Progress Report on Sleeping Sickness in Uganda. By Lt.-Col. David Bruce, F.R.S., R.A.M.C., and David Nabarro, M.D.

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5. Report on Sleeping Sickness from its Clinical Aspects. By GEORGE C. LOW, M.A., M.B., C.M., and ALDO CASTELLANI, M.D. (Florence). Appendix.—*Filaria perstans* and its Relationship to Sleeping Sickness. By GEORGE C. LOW, M.A., M.B., C.M.

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## 3.

# THE DISTRIBUTION OF SLEEPING SICKNESS, FILARIA PERSTANS, &c., IN EAST EQUATORIAL AFRICA.

By CUTHBERT CHRISTY, M.B. and C.M. (Edin.).

[Preliminary Report, dated Entebbe,Ugânda, October 31, 1902.—Received December 5, 1902.*]

(With 3 Maps.)

In studying the Map of the Distribution of Sleeping Sickness, it will be seen at a glance that the disease is connected in some way with the great lake or its waters. In no case has the infection spread far inland, 30 or 40 miles being its limit. It shows no tendency to spread along the Nile source or to other lakes or rivers; 30 or 40 miles, in fact, is beyond the limit. My observations lead me to believe that most cases to be found further than 10 or 15 miles from the lake are cases which have become infected near the shores of the lake. The nearer one approaches the shores of the lake the more prevalent is the disease. Many of the villages by the lake have lost two-thirds of their inhabitants. All the islands in the area are similarly affected. In Buvuma, which I have just recently visited, fully two-thirds of the population have died off, and, at least, half of those now left have the disease; all say, quite cheerfully, that they will get it in time, each day they expect to have the premonitary headache. In this island the mortality has been much aggravated by famine, firstly, the result of drought, and secondly, from dearth of labour, the result of the mortality. The whole island is now almost entirely out of cultivation, except where a few bananas and sweet potatoes are grown by the water's edge. By a cruel irony, one of the most conspicuous symptoms of this mysterious disease in a large number of the cases is an inordinate appetite till the last, the patient continually waking up and asking for food, when he is so weak that he cannot raise himself upon his elbows. It seems pretty certain that the disease is spreading eastwards. Until I visited Southern Kavirondo it was not known to have spread beyond Usoga, but I found it in its worst form all along the lake shore as far as Kisumu, though less prevalent as I neared that place. Beyond Kisumu, and along the south of Kavirondo Bay, I met with no cases, though a very thickly populated district, till I had passed Homa Bay, and reached Kasagunga and Lusinga Island. Here I again met with the disease, some 10 per cent. or more of the population being infected. The configuration of the map of this district would suggest that the disease, after reaching Uyoma promontory in South Kavirondo, has jumped the mouth of Kavirondo Bay and gone on spreading southwards along the east shores of the lake. From information I gathered, it would seem that there is seldom any communication between the people of South Kavirondo and those on the opposite side of the bay. I learnt that, once in a while, Basoga come in canoes along Southern Kavirondo, cross over to Lusinga Island, or to Kissengere, on the mainland, remain there a day or two only, for the purpose of buying salt, and then return again to Usoga. I hope at a future date to again examine the conditions in these districts.

---

* This more or less informal and preliminary report was, as the date shows, written at an early stage of the labours of the Commission. It has been judged desirable to publish it; but Dr. Christy's full report is printed as No. 6 of this series.

The history of the disease probably dates back to the beginning of 1900 in Usoga and Eastern Uganda. In Kavirondo, from what I could gather, the disease was not known earlier than 11 months back, and, in Lusinga Island and Kissingere district, 5 months only seemed to be the duration of the epidemic. This, if correct, clearly points to the direction of spread. The duration of the majority of cases is from 5 to 6 months. The infection would seem to be a "place infection," like that of plague. If a house becomes infected, all the inhabitants usually succumb, and, if others go and live in that house, they also become infected, whereas houses in a shamba only a few yards away remain uninfected, although there is communication between the two; but these points have still to be worked out when the distribution problems are solved. The disease is not, and never has been, very prevalent west of Kampala, and no information has yet reached me of its presence on the western shores of the lake. The assertion that it has spread to the Nandi plateau, as far as I am able to ascertain, is incorrect. I have been unable to trace any connection between the occupation of fishing and the disease. Dr. Hodges' fish theory of causation has, however, points in its favour, and I hope in time to make a careful study of the subject. That the disease exists in West Africa we know, and it is possible that it may have been brought to Usoga from the Congo regions—from the Lendu-Congo districts with the remnants of Emin Pasha's army of Sudanese—but that it has spread gradually across the Continent is not a fact.

If, now, one turns to the Map of the Distribution of *Filaria perstans*, it is seen at once that it in no way agrees, as was supposed, with that of sleeping sickness. As one passes from Usoga into Kavirondo, the percentage of *F. perstans* at once drops from 60 or 70 per cent. to less than 14 per cent., and a little further to 5 per cent. Still further east, beyond the Nzoia River, it is entirely absent, and, in all probability, does not exist elsewhere in the East African Protectorate. Its northern limit, with one exception, is the Usoga boundary, viz., Lakes Choga and Mpologoma, the latter being merely an extensive swamp. The one exception is Mount Elgon. All the slopes of this enormous mole-hill (14,200 feet), except to the north-eastward (uninhabited), are extremely thickly populated. When one's view is not obscured by rainstorms or mist, thousands upon thousands of huts and clearings can be counted dotted about the slopes, which for miles are vivid green, with endless banana plantations, in which the plants grow from 15 to 18 feet high. The Bagesu, the inhabitants of this district on the western and northern slopes of Mount Elgon, are "degraded and simian-like negroes," of small stature and ugly features, which Sir Harry Johnstone says are "perhaps the wildest people to be found anywhere within the limits of the Uganda Protectorate." I did not find them so, for, taking a hint from a neighbouring chief, who sent a message to them explaining who I was, I left my caravan in camp below, and climbed the slopes of the mountain, accompanied only by two interpreters and a porter carrying my blood-testing case. After some patience I was repaid by getting a considerable number of blood slides. I believe few people have ever met the Bagesu of Western Elgon in large numbers on friendly terms, as I did on that occasion. I hope to do so again, for I had no opportunity of examining their dwelling huts, and a knowledge of their domestic life would be of great interest, as possibly throwing some light on the life history of *F. perstans*, for many of the slides taken then contained an enormous number of worms, 100 in one slide being common.

There is no evidence, in my opinion, to point, as has been asserted, to the recent introduction of *F. perstans*, whereas sleeping sickness certainly was, recently introduced, or has recurred after the lapse of many years, of which, however, there is no evidence. The first case was reported in April, 1901, from Kampala, though it probably existed in Usoga previous to that date, being masked by the prevailing famine. The suggestion that sleeping sickness will eventually spread over the *perstans* area cannot be put forward.

In passing [northwards] through Usoga the percentage of *F. perstans* remained high (60 to 80 per cent.) up to the last village (Engo), on the southern shore of Lake Choga. Crossing the lake into Bukedi, the percentage fell to 15 per cent., and two marches further on to 2 per cent. only.

In Bukedi I found from 2 to 4 per cent. of *Filaria diurna* (*nocturna*), and, although the slides containing *F. perstans* were few and far between, all but one of the slides containing *F. diurna* contained also *F. perstans.* Beyond this fact, which, if not a mere coincidence, might point to the probability of the two species being introduced by the same mosquito, I have found no evidence to lead to the belief that the intermediate host of *F. perstans* is a mosquito at all. These few marches through Bukedi, where I touched an area of *Filaria diurna*, which probably extends as far as Egypt, were the only marches during which mosquitoes (a large straw-coloured species, with conspicuously black-banded legs) were a torment, biting all day in thousands, and necessitating a wood fire by the dinner table in the evening.

The distribution map of *F. perstans* is compiled from information gained from blood slides other than those taken on my march through the country.

There used to be great enmity between the Usoga and the surrounding tribes, but there is now free intercourse, so that it is possible that *F. perstans* has only recently extended beyond the borders into Bukedi and Kavirondo by persons who have become infected when visiting Usoga.

On Mount Elgon, although blood slides have been obtained from only one place amongst the Bagesu, I have marked what I think is their area of occupation as equally infected with *F. perstans* as that from which I obtained the slides, as the conditions seemed to be the same. Although I have examined a very large number of blood-slides, I have failed to find *F. perstans* in the blood of sleeping sickness cases living beyond the area marked as that of *F. perstans*; and in the area of both, if a sufficient number of slides are examined, the percentage of *F. perstans* in the sleeping sickness cases always agrees with that of *F. perstans* in the healthy populations.

Southern Kavirondo is an extremely fertile country; the inhabitants are more or less industrious, and enormous quantities of grain are grown, which supply the needs of a vast area to the east and north-east. In passing from this grain country across the border into Usoga the traveller, after crossing the mile strip of no-man's-land, is at once struck by the changed surroundings. In place of the cornfields he finds bright green banana plantations and areas of dense forest with large trees.

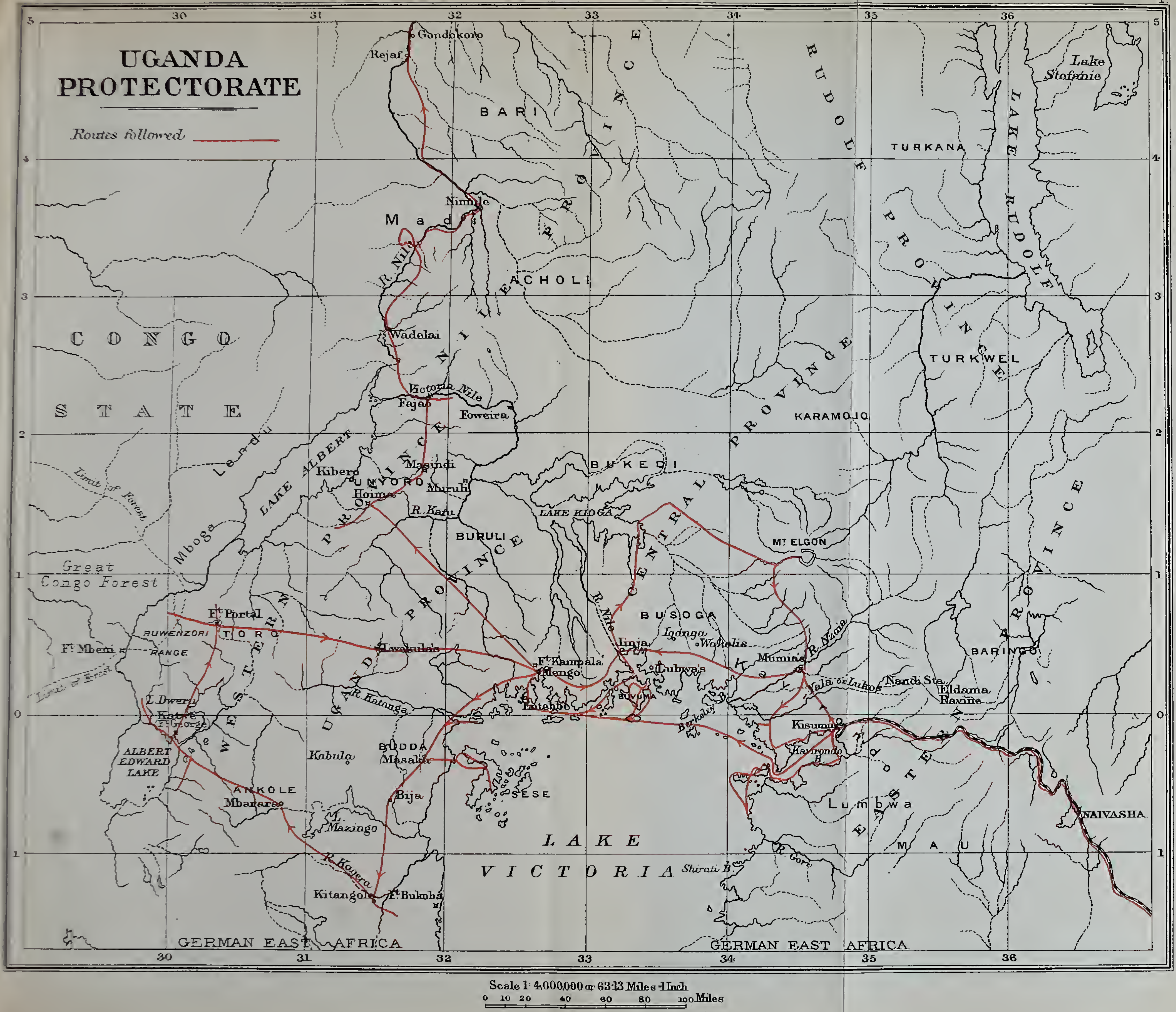
Upon studying those boundaries of *F. perstans* as yet mapped out and comparing them with the boundaries of the banana cultivation, it will be seen that the two coincide in a remarkable manner.

This fact must be something more than a coincidence. Wherever the banana is growing, in the districts I have examined, there is found the *F. perstans*. What is more, it would seem that where the banana is most luxuriant (on Mount Elgon) there the inhabitants are most infected (60 to 70 per cent. and the blood crowded). The converse of this is also clearly correct, as will be seen by the map, viz., where the banana is scarce (in Southern Bukedi, and Northern Kavirondo, in which latter place it is only grown inside the mud walls of the small villages) there the percentage of *F. perstans* is small; and where there is no banana there is no *F. perstans*. The banana, I am told, is grown all over Uganda and the Western Provinces (Ankole, Toro, and Unyoro), its limit to the north being the Victoria Nile. It remains for me to prove, if this is so, whether the Nile is here also the limit of *F. perstans*. From slides I have obtained from these districts this would seem to be the case. The banana when fully grown is peeled and boiled or steamed in an earthenware pot. The leaves are used for plates and dishes, for thatching houses, for clothing, for mattresses to sleep upon, for pipe stems and many other things. The skins of the fruit are used for making soap, and the stems for rope, etc.

There is yet another area which coincides in almost as remarkable a manner with that of *F. perstans* as does the area of cultivation of the banana. This is the area in which live people who wear clothes. To the north and east of the red pencil line on the banana map are the Nilotic races to the north of the Victoria Nile, the Bukedi (the word meaning "the land of naked people"), the Bagesu on Mount Elgon (some of whom, however, as I saw them wore small pieces of bark-cloth and skins), and the Kavirondo, who, like the Bukedi, are, both men and women, entirely naked, not even using a loin-cloth. When coming into Kisumu market a small goat-skin, in the case of the men, is occasionally worn over the buttocks, but nothing in front, while some of the women wear a few red and white beads in front and a tassel of string or grass behind. The line of demarcation between the clothed and unclothed is everywhere distinct, but as "civilisation" advances this line will gradually fade with increasing rapidity, as it is now said to be doing. The Victoria Nile therefore has a treble interest attached to it, for it remains to be discovered whether it is the limit of the areas of *F. perstans*, of the banana, and of the clothed people, in a similar manner to that already shown to be the case to the eastward, and mapped out in the attached maps. The word clothing has not, of course, the significance that it has in Europe. It here means merely a loose gown of white Americana, a toga of brown or black native bark cloth (hammered out from the bark of a tree), a specimen of which is enclosed, or a small piece of bark-cloth suspended from the hips. Working in the fields the cloth, in most cases, is discarded.

With regard to mosquitoes, I have made a careful collection all along my route. These are now in the hands of Mr. Theobald of the British Museum (Natural History). There does not seem to be any one species which prevails generally in the *F. perstans* area. The commonest species (? a Stegomyia) was equally common in parts of Kavirondo. The greater part of Kavirondo is high land and open, and the temperature is cool. Mosquitoes therefore at many camps were absent. In no part of Kavirondo, as far as I saw, are mosquitoes a trouble. In Usoga they are more troublesome but nowhere a pest, unless it be as one nears the big swamps surrounding Lake Choga. At most places along the shores of the Victoria Nyanza and in the islands they are seldom noticeable. After crossing Lake Choga into Bukedi they at once become as great a pest as they are on the Niger in West Africa and elsewhere. In the swamps and long grass of Bukedi they are intolerable all day long, and towards evening wood fires are required, the smoke of which is almost as intolerable as the mosquitoes. As one passes eastwards towards Mount Elgon they again become scarce, and on Mount Elgon itself I collected none.

The traveller in Usoga continually has thrust upon his attention pests worse than mosquitoes, viz., bed bugs and body lice. The native of Usoga carries everywhere on his person or clothes a large supply of both, and no amount of ingenuity will prevent the animals from getting from the porter into the load he is carrying. A good supply can always be obtained by searching his piece of bark-cloth, and if more are required hundreds can be found in his hut ensconced in the folds of his papyrus or banana-leaf mattress and elsewhere. In Kavirondo with Kavirondo porters the traveller is free from this annoyance, at least I was, and others I questioned on the subject also had been. The Kavirondo has no clothing in which to harbour the pests, nor has the man of Bukedi. From the few observations I have made, their huts would also seem, for some reason, to be either quite free or less infested than those of the Busoga, but as yet I can make no definite assertion upon this point. The Kavirondo, at all events, make freer use of the universal ammoniacal insecticide—cow-dung—so freely used in West Africa and all over India and other parts of Asia and elsewhere.

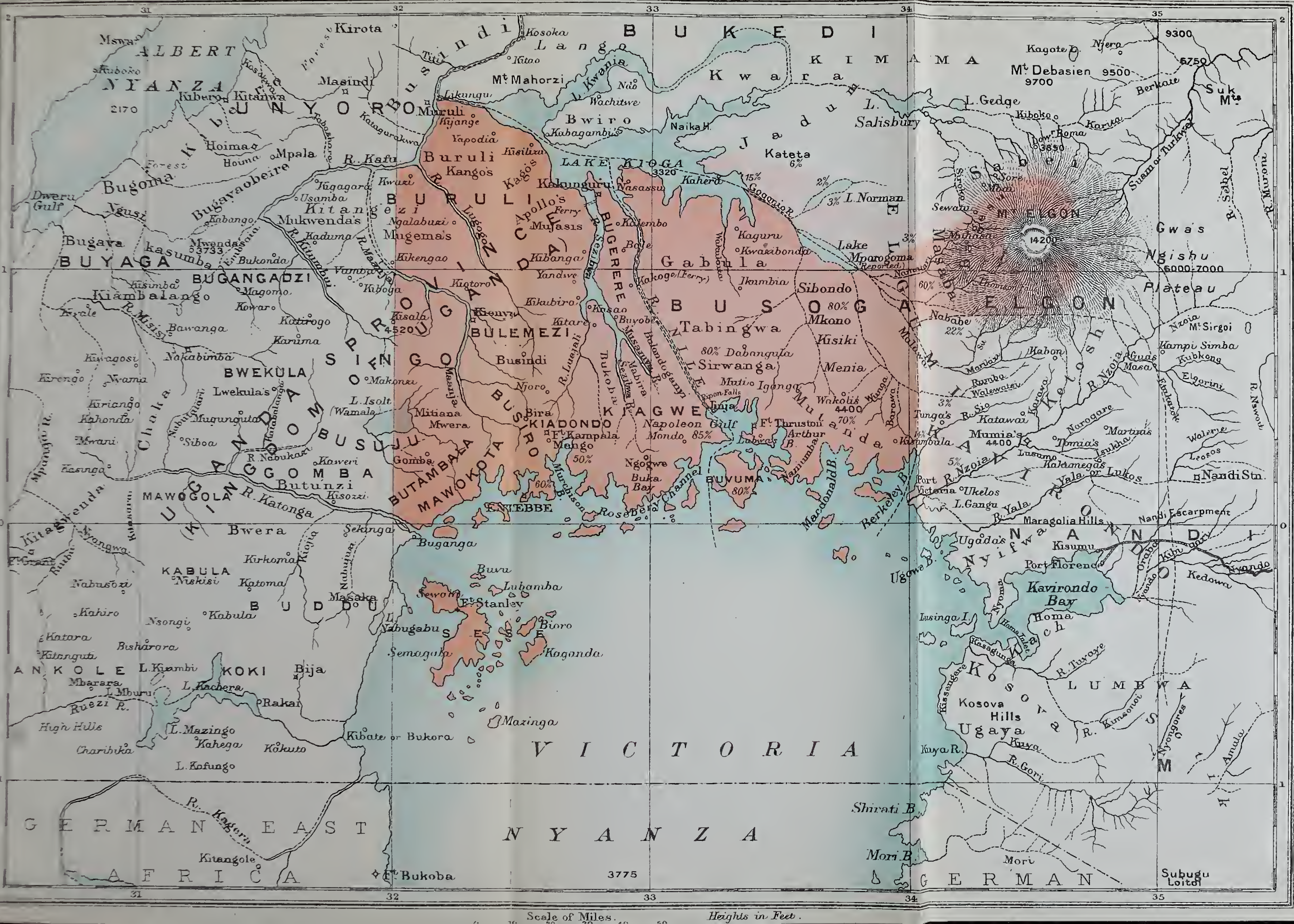


# UGANDA PROTECTORATE

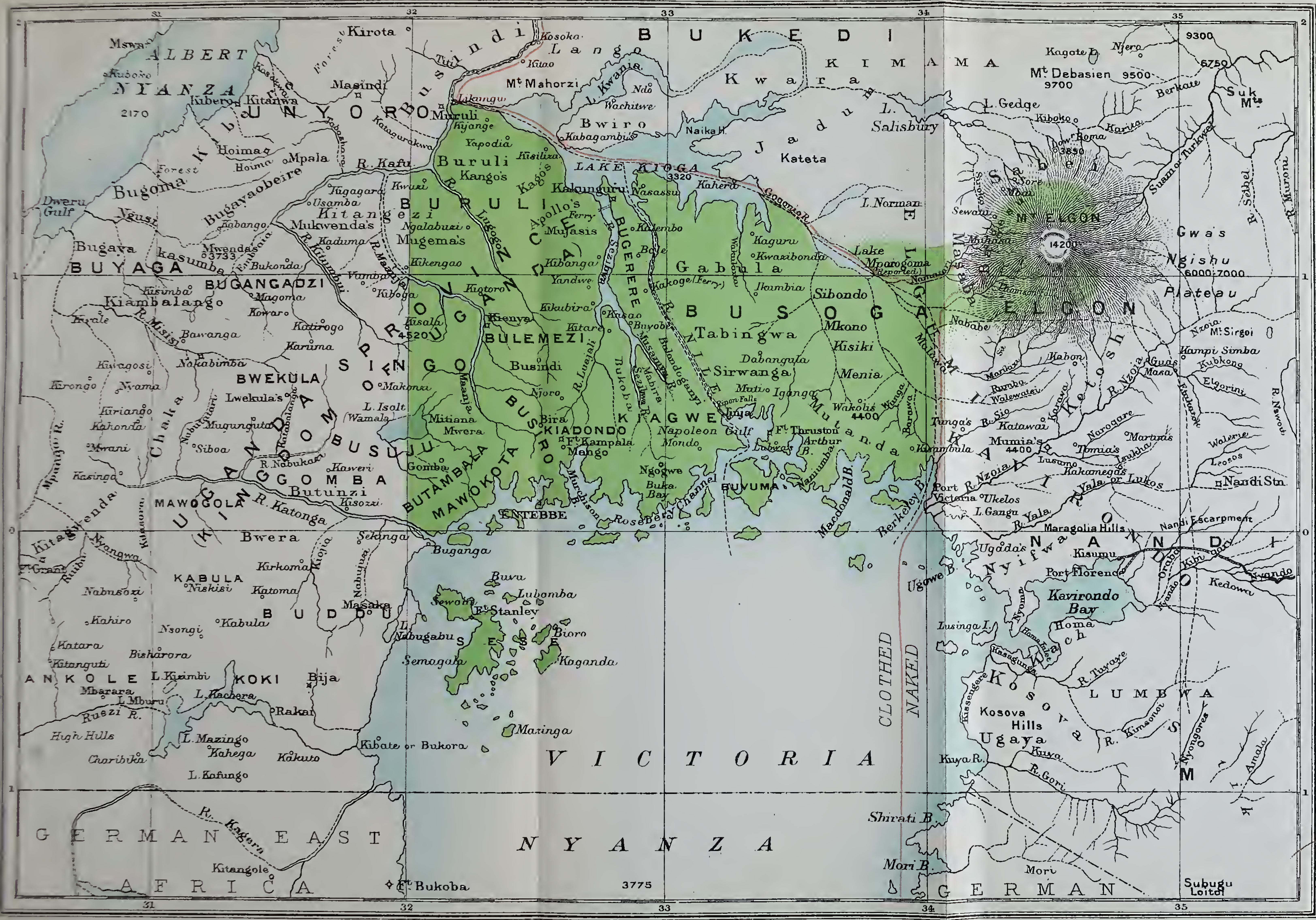
Scale 1: 4,000,000 or 63·13 Miles = 1 Inch

## DISTRIBUTION OF FILARIA PERSTANS AS KNOWN UP TO DATE OCT. 23RD 1902.

II



DISTRIBUTION OF THE BANANA CULTIVATION AND CLOTHED PEOPLE. III.



Scale of Miles. 0 10 20 30 40 50 Heights in Feet.

## 4.

# ADULT FORMS AND DEVELOPMENTAL FORMS OF THE TRYPANOSOME FOUND IN SLEEPING SICKNESS.

By ALDO CASTELLANI, M.D. (Florence), (Professor of Pathology and Bacteriology, Colombo Medical College).

[Received July 21, 1903.]

[With 2 Plates.]

In previous publications of mine* I stated that in Sleeping Sickness patients I found, in twenty cases out of thirty-four, a trypanosome, which I considered the cause of the disease. My results have been since then amply confirmed by such an authority on trypanosomes as Colonel Bruce. In this note I propose to give some details on the morphology of the adult parasites and their developmental forms.

### *Appearances of the Adult Trypanosome.*

*Fresh Preparations.*—The parasite has the usual general outline of the other trypanosomes. It has a worm-like shape, and one observes in it one end terminating with a flagellum, the other more or less bluntly conical, though sometimes it may be quite pointed, an undulating membrane and a vacuole. The protoplasm does not appear to have quite a uniform structure, but rather an alveolar one, as described by Plimmer and Bradford in Tryp. Brucei, although apparently far from being so well marked.

At first the parasite moves fairly actively, but, on observing the preparation for some time with the microscope, one sees that the movements by-and-by become more sluggish, until they stop altogether. Frequently the trypanosome stops near a leucocyte, which by degrees engulfs it. In other instances, after having slowed down very much in their movements, the parasites stop far from any leucocytes and disappear suddenly as if they had been dissolved by the liquid. On several occasions one sees in fresh preparations trypanosomes with apparently two well-marked flagella; they certainly are parasites in longitudinal division.

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* 'Proc. Roy. Soc.,' May, 1903; 'Journal Trop. Med.,' June 1, 1903.

*Locomotion.*—The trypanosome found in Sleeping Sickness, according to my experience, moves always with the so-called posterior end (blunted extremity) in front, whereas the other known trypanosomes move generally with the anterior end (flagellum) in front. Tryp. Gambiense, for instance, according to the description of Dutton, is usually seen progressing with the flagellum (anterior end) in front, unless there is some insurmountable obstruction, when at times it shoots backwards for a short distance with the blunted end in front.

*Vitality of the Parasite outside the Body.*—The trypanosome may remain alive in fresh preparations of blood rinsed with vaseline for four to six hours. The vitality seems to be longer in the cerebro-spinal fluid, where it may remain alive for fifteen to eighteen hours At this time the number of the parasites is very much decreased and the movements of the few parasites left are very slow. After twenty hours no parasites are visible. Differences in temperature, at least to a certain degree, do not seem to affect much the vitality of the parasite. For this purpose some tubes of cerebro-spinal fluid were kept at the temperature of the room (28° C.), others incubated at blood heat, and others incubated at 18° C. by means of the cooling incubator. The results were alike for all the tubes, viz., some parasites still alive after eighteen hours, and no more parasites present after twenty hours.

*Stained Specimens.*—For staining the parasite, Leishman's modification of the Romanowsky's method gives very good results. Staining the trypanosome in this way, the macro-nucleus, micro-nucleus and flagellum appear red, the protoplasm blue, while undulant membrane remains almost unstained.

The nucleus is generally large and of variable shape. It is as a rule situated in the posterior half of the parasite.

The micro-nucleus (Plimmer and Bradford), or centrosoma (Laveran and Mesnil), does not show apparently any structure. It stains red, but of a much more vivid colour than the nucleus. It is situated very near the posterior end of the parasite, and generally outside the vacuole.

The vacuole is oval and of rather large dimensions. It is situated anterior to the micro-nucleus.

The flagellum takes origin apparently from the micro-nucleus, then following the external edge of the undulating membrane, reaches the anterior extremity, where it becomes free. The free portion of the flagellum is usually longer than in other trypanosomes.

The protoplasm does not stain evenly and not very deeply; it does show some chromatic granules.

The total length of the parasite is from 16 to 24 $\mu$. The width is from 2 to 2·5 $\mu$

The adult forms of the parasites are shown in Figs. 1—5.

### *Atypical Adult Forms.* (Figs. 6, 7, 8.)

Besides the adult forms already described, one meets with other rarer forms with different shape. The parasite has lost its slender outline and has become thickened, in fact almost sausage-like. The posterior end is much more rounded, the free portion of the flagellum is shorter. The vacuolum may take greater dimensions. The protoplasm is less well stained. Round the nucleus there may be several points where the chromatine collects. In analogy to what Laveran and Mesnil have observed in Trypanosoma Lewisi, I am inclined to think that these atypical adult forms are parasites which prepare for division. These forms may be observed in the blood as well as in the cerebro-spinal fluid.

### *Forms in different Stages of Longitudinal Division.* (Figs. 9, 10, 11.)

The very first stages of this mode of division are probably represented by the atypical adult forms already described. Then the division begins probably from the micro-nucleus. Forms as represented in figs. 9 and 10 are fairly frequent; the division has gone so far as the micro-nucleus and portion of the intra-body part of the flagellum, but no signs of the division of the nucleus and protoplasm has yet appeared. Fig. 11 shows a trypanosome in which the division is almost complete. The two new parasites are only kept united by the posterior end. These forms may be seen frequently enough in fresh preparations moving about very actively.

### *Various Types of Development Forms.*

Group 1 (Figs. 12, 13, 14).—In the blood taken from the finger, especially during the last stages of the disease, in the blood of the heart, as well as in the cerebro-spinal fluid, I observed frequently special roundish bodies of about 10 to 14 $\mu$ in diameter. Their protoplasm is very finely granular, and one can see in it one or more vacuola. These bodies on a very superficial observation might be taken for amœbæ. This idea of course cannot be entertained, because they never emit pseudopoda. These bodies may change very slowly, their general shape passing, for instance, from a round form to an ovoid one. This change of shape takes place in a long time. I could never follow these bodies long enough to see their ultimate transformation. Stained with the Romanowsky-Leishman method, they show several points where the chromatine collects, and, sometimes, very fine flagella. Sometimes there is only one large vacuole, in other instances there may be several vacuola. Similar bodies have been described by Rabinovitsh and Kempner in Trypanosoma Lewisi. Rabinovitsh and Kempner have drawn them without flagella, but they admit now* that possibly some very fine flagella may be present. The interpretation of these bodies is very difficult; they might be degenerative forms, but I am more inclined to consider them developmental forms of the parasite. Doflein thinks that the Rabinovitsh-Kempner bodies observed in Tryp. Lewisi represent perhaps stages of a sexual reproduction.

Group 2. Amœboid forms. (Figs. 15—19.)—These forms were first described by Plimmer and Bradford in Tryp. Brucei. They are very small, their diameter being 5 to 7 μ; the shape is variable. The typical ones are more or less pear-shaped. At the acute extremity there is very frequently a fine short flagellum. A macro-nucleus and a micro-nucleus are present; sometimes they may be divided. Round the macro- and micro-nucleus there is an unstained portion like a vacuolum. The flagellum takes origin apparently from the micro-nucleus. The amœboid forms I have seen only in the cerebro-spinal fluid. Sometimes one observes a large number of these amœboid forms agglomerate together something like a plasmodium formation. (Fig. 22.)

I do not intend to go into the question as to what these bodies really represent; it is well known how far the opinions of Plimmer and Bradford, and Laveran and Mesnil differ on the amœboid forms of Tryp. Brucei, but I think it is interesting to note that identical bodies are seen also in the trypanosome of Sleeping Sickness.

### *Investigation of Tissues for Trypanosomes.*

In animals infected with Nagana or Surra the parasites may be found in some organs,-spleen, &c. Very interesting are the experiments of Plimmer and Bradford, who observed the capillaries of brain of animals infected with Nagana full of amœboid forms. Mr. Plimmer has very kindly examined pieces of brain and spleen from Sleeping Sickness patients. So far he has not been able to find any parasites. I hear also that Professor Mott has not yet found parasites in the tissues he has received from Uganda. I have also examined several pieces of tissues of brain and spleen, hardened in alcohol embedded in paraffin and stained with Leishman's method, but so far I have found nothing. Still, too much importance must not be attached to these negative results, as this investigation has been carried out on sections of the tissues. Smears of the tissues might give much better results.

From my researches it would seem that in the trypanosome of

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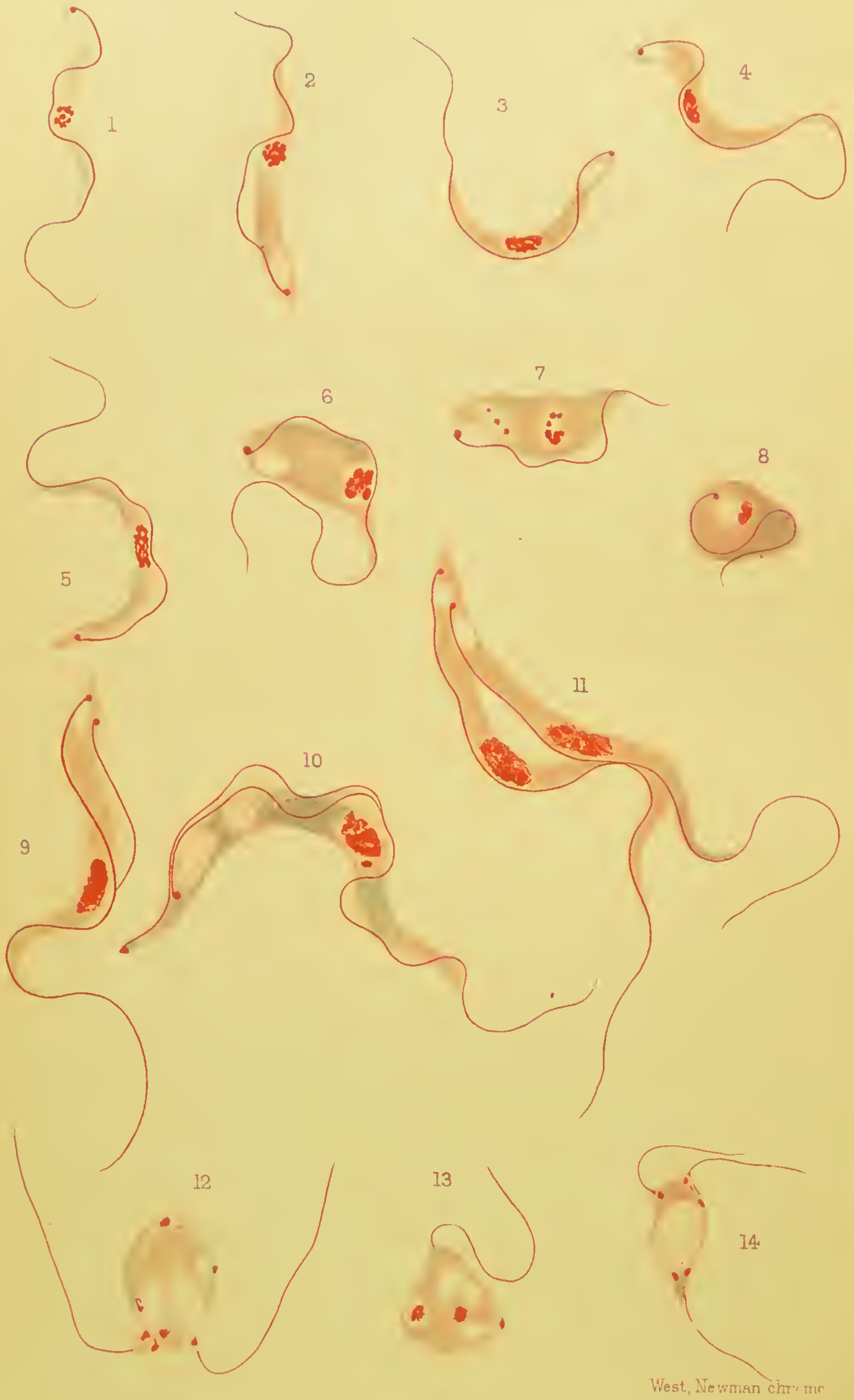
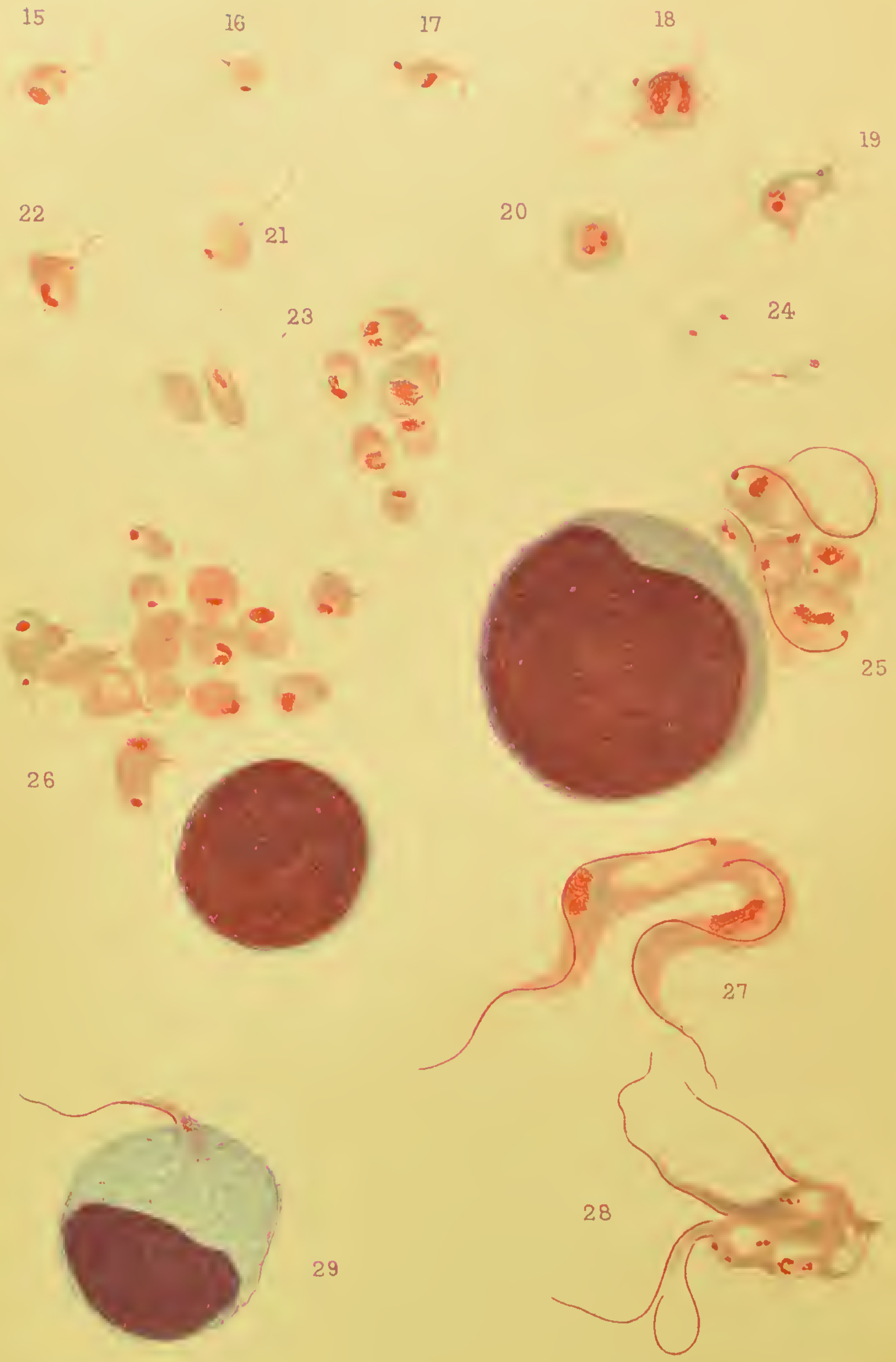
* Oral communication.

*Castellani.* *Trypanosome of Sleeping Sickness, Pl. I.*



West, Newman chromo



West, Newman chromo

Sleeping Sickness one must distinguish morphologically several forms: typical adult forms; atypical adult forms; forms in different stages of longitudinal division; Rabinovitsh-Kempner bodies; Plimmer-Bradford amœboid forms. It would be premature to come to any definite conclusions, but from these observations it would seem that in the trypanosome of Sleeping Sickness the multiplication by longitudinal division is not the only mode of reproduction.

## DESCRIPTION OF PLATES.

### Plate 1.

Figs. 1—5.—Typical adult forms.

Figs. 6, 7, 8.—Atypical adult forms.

Figs. 9, 10, 11.—Forms in various stages of longitudinal division.

Figs. 12, 13, 14.—Different types of developmental forms.

### Plate 2.

Figs. 15—19.—Amœboid forms (cerebro-spinal fluid).

Fig. 20.—Aggregation of amœboid forms near a leucocyte (cerebro-spinal fluid).

Fig. 21.—Fusion form? This is probably a degeneration form representing perhaps several trypanosomes which have conglomerated and fused.

Fig. 22.—Aggregation of amœboid forms simulating perhaps a plasmodum formation (cerebro-spinal fluid).

Fig. 23.—Amœboid and fusion forms.

Fig. 24.—Conjugation form? (cerebro-spinal fluid).

Figs. 25—26.—Aggregation of amœboid forms.

Fig. 27.—Conjugation form.

Fig. 28.—Fusion form? This represents, perhaps, the several trypanosomes which have fused together, and are in a state of degeneration.

Fig. 29.—Leucocyte engulfing a trypanosome.

## 5.

# REPORT ON SLEEPING SICKNESS FROM ITS CLINICAL ASPECTS.

By GEORGE C. LOW, M.A., M.B., C.M. (Superintendent London School of Tropical Medicine),

AND

ALDO CASTELLANI, M.D. (Florence), (Professor of Pathology and Bacteriology, Colombo Medical College).

[Written June, 1903.—Received August 4, 1903.]

The following data were collected in the months of July, August, and September, 1902, in the special hospital erected by the Government, in Entebbe, for the study of Sleeping Sickness.

### *Synonyms.*

This disease is known by many different names:—By the natives of Africa in Uganda as Mongota (To nod). On the Congo as Yela Kwa Tula, Manungina, Lalangolo, N'tansi or N'tola; Nelavane (of the Wolofs), Dadane (of the Sereres), Toruahebue (of the Mendehs).

By European observers it is known as Sleeping Sickness, Sleeping Dropsy, Negro Lethargy, African Lethargy, Die Schlafkrankheit der Neger, Malattia del Sonno, Doenca de Somno, Enfermedad del sueno, Maladie du sommeil, Maladie des dormeurs. It might perhaps be more correctly termed African meningo-encephalitis, or shortly, African Meningitis.

### *History.*

The first to observe this disease was apparently Winterbottom, who wrote a short description of it in a paper entitled "An account of Native Africans in the neighbourhood of Sierra Leone" in 1803. For many years after this little if anything seems to have been written on the subject till 1868, when Dumontier and Santelli both wrote independent papers on the disease. Another paper, by Guerin, appeared in the following year, but it was not until Corre studied the disease amongst the natives of Senegambia, that a complete and fairly accurate account of this malady was given.

It has been stated that Sleeping Sickness has been seen in Negro slaves imported to the West Indies and in Brazil, but the evidence of this is rather fragmentary, and never appears to have been directly proved. In 1891 Mackenzie published the record of a case which was under his care in the London Hospital, and in 1900 Manson gave a very complete description of two cases in Charing Cross Hospital sent from the Congo by Dr. Grattan Guinness. The pathology of these two cases was very thoroughly worked out by Mott, who first accurately described the pathological histology, and pointed out that the lesion was one of the nature of a meningo-encephalitis. More recently the disease has become more common in different parts of Africa, and much attention has been paid to it in Senegambia by Marchoux and Dantec; in Portuguese West Africa by Cagigal and La Pierre, and also by the Portuguese Commission; in the Congo by Brodin; in Uganda by Cook, Moffat and Hodges.

### *Geographical Distribution.*

The endemic area of Sleeping Sickness is limited to parts of Equatorial Africa.

Until a few years ago the geographical distribution of Sleeping Sickness was limited to West Africa from Senegal in the north to San Paolo de Loanda in the south. It has also been known for many years on the Congo, and lately has spread up that river to beyond Stanley Falls, and it is probably much more common than supposed in hinterlands of most of the West Coast areas between the abovementioned limits.

On some areas on the Upper and Lower Congo the disease has lately been seen as an epidemic which has killed off large numbers of the population.

In 1900 Cook first discovered that Sleeping Sickness existed in Uganda, and since that date the disease has spread widely, keeping, however, more or less limited to the north shore of the Victoria Nyanza Lake. The epidemic seemed to start in Usoga, and from this centre has spread to Buddu in the west, and down to Kisumu, the rail head in the east. Northwards Cook has recorded cases from Kiadondo, but it does not seem to have spread much in this direction, as it is unknown in some of the northern provinces, in Unyoro and all the districts about the Albert Lake, the same applying to the Nile districts. Its centre of virulence has been in Usoga, in Chagwe, around Entebbe, the seat of the Government, and also in all the islands adjoining the mainland, from the Sese group in the west to the Kavirondo Islands in the east, some of these latter areas having been decimated by this scourge. It has not been reported in German East Africa to the south and east of the lake, and it is unknown in the Nandi and Masai tribes who inhabit the territories of British East Africa east of Kisumu.

## *Etiology.*

The most different hypotheses have been brought forward to explain the genesis of the disease. Some old observers thought that the disease may have originated from emotional distress connected with negro slavery. Other authors considered the disease a form of sunstroke or a variety of beri-beri or malaria. The disease has also been attributed to scrofula. More recent theories have connected Sleeping Sickness with an intoxication of food, animal parasites, and with bacteria.

*Food Intoxication.*—Some Portuguese observers, and more recently, Ziemann, have regarded Sleeping Sickness as a sort of intoxication. Ziemann thinks it a manioc-intoxication and compares the disease to Pellagra, which by most authors is also considered an intoxication from food (maize). Against this theory of Ziemann's is the fact that in Uganda Sleeping Sickness has appeared only in the last 3 years, and no change of food whatever has taken place. The staple food of the Baganda is now, as it was before, bananas, and among the tribes near the lake also a little fish.

*Parasites. Filaria perstans.*—Manson, remarking the singular correspondence between the distribution of the disease and that of *Filaria perstans*, suggested that this parasite might in some way be responsible for Sleeping Sickness. There is no doubt that this theory has some strong points in its favour It would explain well the long incubation period of the disease. Sleeping Sickness, it is said, may develop in negroes many years after they have left the endemic area, and it is well-known that *Filaria perstans* may remain alive for years. This theory has been disproved by the observations of Low, who has shown that in British Guiana, where this parasite is common, there is no Sleeping Sickness, and *vice versâ* in Kavirondo, where the latter disease is rife, there are no filariæ.

*Rhabdonema Strongyloides.*—The embryo of this worm was considered by Forbes as the cause of Sleeping Sickness. This parasite, according to Tessier, penetrates the mucous membrane of the intestines and reaches the general circulation, and is then retained in the cerebral vessels.

*Ankylostoma Duodenale.*—Fergusson considers this worm as the cause of the disease. In Uganda this parasite is met with very frequently in Sleeping Sickness, but it cannot be considered the cause of the disease, as the administration of an appropriate anthelmintic frees the patients from it, and sometimes improves the general conditions for a few days, but the fatal course of the disease is not checked.

*Bacteria.*—Cagigal and Lepierre in 1897 announced that they had isolated a bacillus from the blood of a patient suffering from Sleeping Sickness, which when injected into animals was able to reproduce the disease; but these experiments were not confirmed later by Brault and Lapin. Next, Marchoux, basing his statements on the following observations, is of the opinion that Sleeping Sickness is due to Fränkel's diplococcus. At the *post-mortem* examination in a case of Sleeping Sickness, where the patient was suffering also from pericarditis, he found Fränkel's diplococcus in the exudation of the pericardium. In a second case, complicated by chronic rhinitis and suppuration in the sinus frontalis, the secretion of the nose showed the same micro-organism. Besides these two observations, to which much importance cannot be attached, Marchoux was brought to his hypotheses by having noticed that Sleeping Sickness sometimes developed in people who had suffered from pneumonia. Lately there have been the bacteriological researches of the Portuguese Commission, and those of Dr. Broeden. The Portuguese describe a diplostreptococcus, which they state they have found constantly in the cerebro-spinal fluid at the *post-mortem* examination of their cases. They state that they have frequently found the same organism in patients during life in cerebro-spinal fluid taken by lumbar puncture. According to their description, the characteristic feature of this germ is that it does not grow at all on gelatine and very poorly on the other usual culture media.*

Dr. Broden, of the Bacteriological Laboratory at Leopoldville, describes as the cause of the disease a bacillus which is slightly motile, producing a pellicle on bouillon and growing abundantly on potatoes. He was able to find the germ constantly in the blood of all his patients. The bacillus was not agglutinated by the blood of patients suffering from Sleeping Sickness.

In non-complicated cases Castellani has found frequently a streptococcus. Out of thirty-nine cases, he has grown streptococci from the blood of the heart in thirty-two, and from the liquid of the lateral ventricles in thirty. The germ does not seem to be present frequently in the organs. He has found it a few times in microscopical sections of the brain and spleen. During life he found the germ very rarely, and only in the last stages of the disease. From the blood taken from a vein he grew it once only, although he examined bacteriologically the blood of thirty-seven patients, repeating the investigation several times and with different methods. He examined the cerebro-spinal fluid obtained by lumbar puncture in twenty-eight patients; in five he had positive results, but only a few hours before death, with the exception of one case, in which he found the streptococcus several days before death. In six bacteriological examinations of the urine he grew the germ once; this was in the same case in which it was found in the blood.

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* In a recent communication the members of the Portuguese Commission have greatly modified the description of their organism, stating that it also grows on gelatine and on the other usual culture-media.

In three patients he punctured the spleen during life; the bacteriological examination of the spleen juice, obtained in this way, always gave negative results. The bacteriological examinations of enlarged lymphatic glands, removed during life, was negative.

*Trypanosoma.*—Since November, 1902, when he began to use a special technique, Castellani observed very frequently a trypanosoma in Sleeping Sickness patients. He found this trypanosoma in the cerebro-spinal fluid of twenty out of thirty-four patients. In the blood, among the few cases examined, he found it in one, and in several cases he observed special bodies which he considers developmental forms of the parasite. These bodies can be seen also in the cerebro-spinal fluid. In this fluid may be present also other bodies very small, 4—6 $\mu$ in diameter, which, when stained with Romanowsky's method, show a macro-nucleus and a micro-nucleus, from which a flagellum takes origin. These bodies are identical with the amœboid forms described by Plimmer and Bradford in Tryp. Brucei.

### *Predisposing Causes.*

So far, the disease has been observed in negroes only. A case was observed by Chassaniol in a mulatto, and one by Clarke in a creole boy. It is said to occur in negroes long after they have left the endemic area. Sex does not exercise any influence, neither does age. The disease is equally frequent among children, young and old people. As important predisposing causes, Clarke mentions disorders of circulation, mental depression, bad and insufficient food. It may also be that parasites like filaria perstans, ankylostoma duodenale, etc., so common in natives, impairing the natural forces of resistance, may play a certain rôle as predisposing causes in the ætiology of the disease.

## SYMPTOMS AND CLINICAL FEATURES OF SLEEPING SICKNESS.

### GENERAL SKETCH.

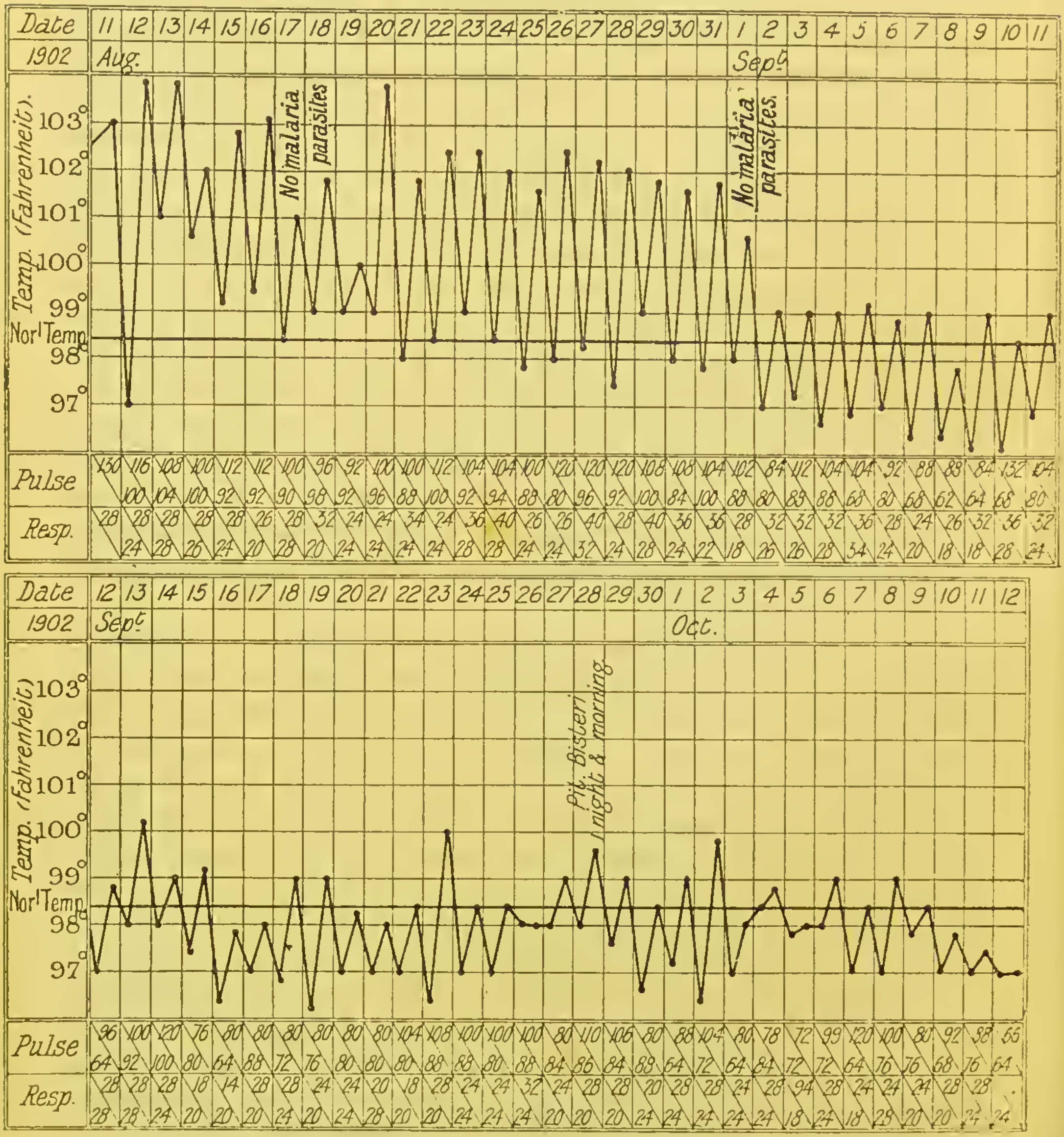
The symptoms of the disease begin very insidiously, some slight change in the former mental attitude of the patient being the first thing noticed by the relatives of the patient. Next a disinclination to work, with a tendency to sit about and rest more than usual, appears, and at this time headaches and other transient pains may be complained of, especially pains in the upper part of the chest. The facial aspect now also changes and a previously happy and intelligent looking negro becomes instead, dull, heavy and apathetic. Once those changes have appeared, the disease may run an acute or more or less chronic course, progressing however to its ultimate fatal termination. It is about this time that one usually sees the case, and an ordinary inspection will reveal many of the following points:—There is the dull, heavy, stupid look, a slowness in answering questions, and when speech does come it is often mumbling, slow and thick; the gait is best expressed by the term shuffling. Headaches, vague pains and chest pains may be complained of. The tongue may or may not at this time show the characteristic fine tremor, and in some cases this may also be noticeable in the hands. The skin is often soft and smooth, or it may be slightly roughened. Glandular enlargements common amongst all natives may be prominent, but in some cases this may be very slight. The temperature—a very important point—is elevated, rising in the evenings to 101° or 102° F., falling to subnormal in the morning, the range often extending over 4° or more, and the pulse of very low tension is accelerated, varying from 90—130 beats per minute. These two symptoms are of the greatest diagnostic importance in the early recognition of the disease. On interrupting the examination and quietly watching the patient, he will probably sit down, his head may nod, his eyes close, and he remains in this drowsy lethargic condition until again asked some question. If one take such a case into hospital, for the first few days a slight improvement may take place; the patient gets up from his bed daily, sits about the doors of the hospital, sometimes walks about outside and takes a little more interest in life, especially at meal times. Soon, however, depending on whether the disease is to run an acute or chronic course, the individual gets worse, he stays in bed more, becomes more drowsy and lethargic, though not actually sleeping, walking at the same time becomes more difficult, and he eventually remains constantly in bed.

Tremors now usually become marked, these being of a fine nature. They are best seen in the tongue and arms. The skin may become rough and lose its lustre, but eruptions, though they have been described, are not common. Emaciation and general weakness becomes pronounced; the knee reflexes, which were at first somewhat exaggerated, become diminished, the motions are passed involuntarily in bed, and saliva often dribbles from the mouth. Drowsiness, which has gradually been increasing, now passes on to coma, from which the patients can only be roused with difficulty; the temperature falls to subnormal, in rare cases convulsive fits appear, and the patient dies in a complete state of coma. This is the common course of an ordinary acute case of the disease, the different changes taking about a month or six weeks for completion. In the chronic cases the symptoms develop more slowly, and they remain more constant for considerable periods of time without any advance, but ultimately the patients pass into the later stages described above and eventually die.

## *Analysis of Symptoms.*

*The Temperature. Regular Course.*—The course of the temperature is marked in typical cases by an evening rise and a morning fall, the range often extending over 4° or more. The night temperature varies from 101°, 102° to 103° F., and the morning temperature is generally about the normal, sometimes, however, a little over this or often distinctly subnormal.

Charts 1 to 5.—Typical Temperature Charts.



Pil. Bisleri = Iron, arsenic and quinine.

Chart 2.

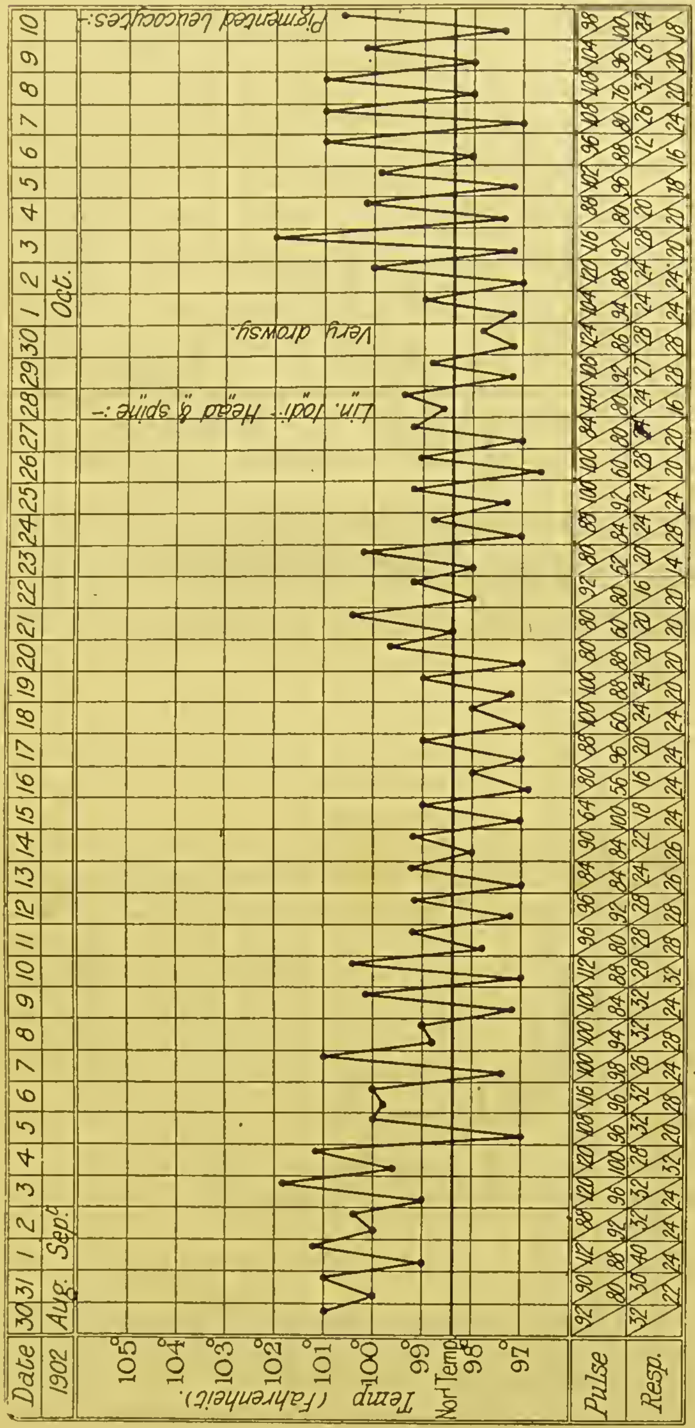

Date
1902
Aug.
Sep.
Oct.
Temp (Fahrenheit).
105°
104°
103°
102°
101°
100°
99°
Nor Temp.
98°
97°
Pulse
Resp.
Lin. Iodi: Head & spine:-
Very drowsy.
Pigmented leucocytes:-

Chart 3.

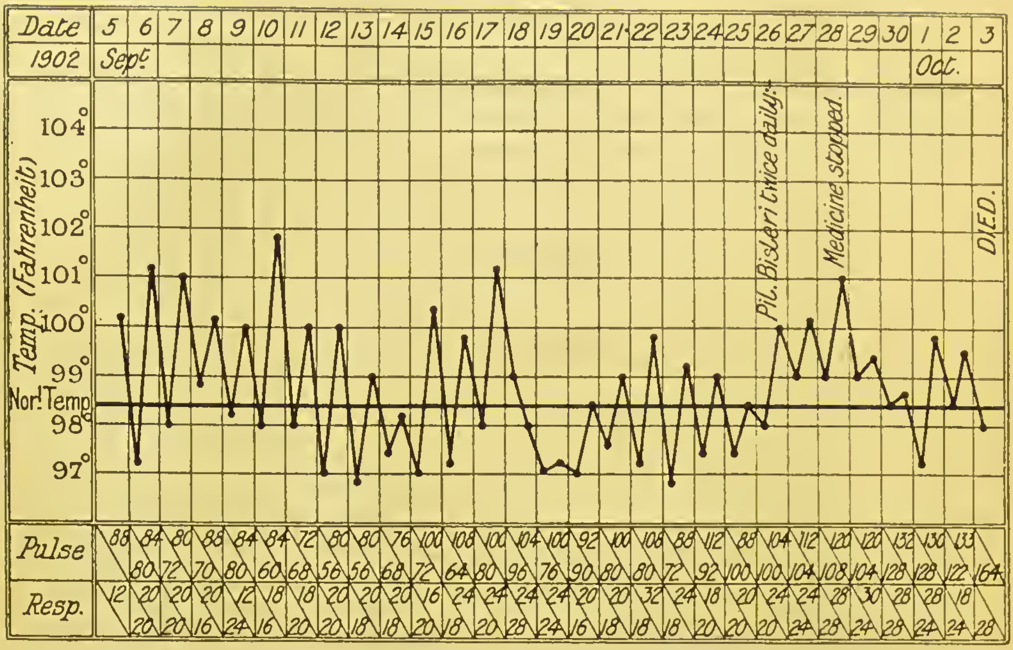


Chart 4.

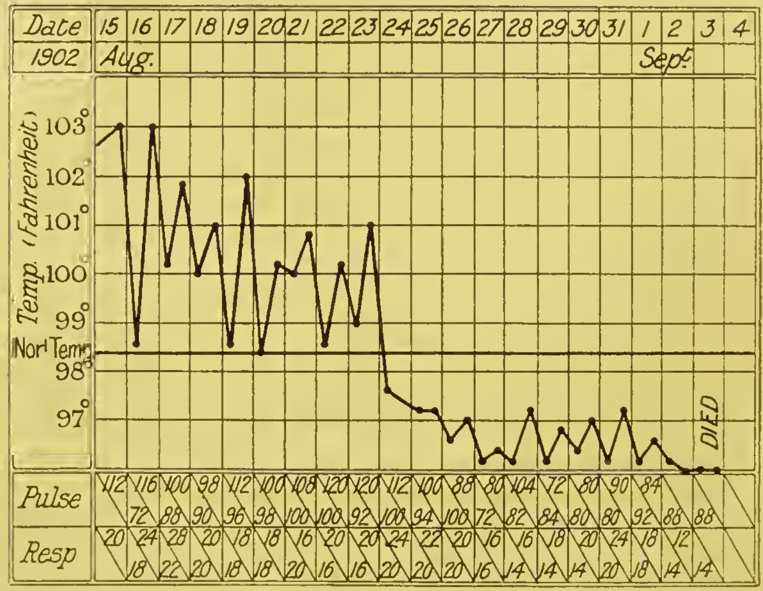
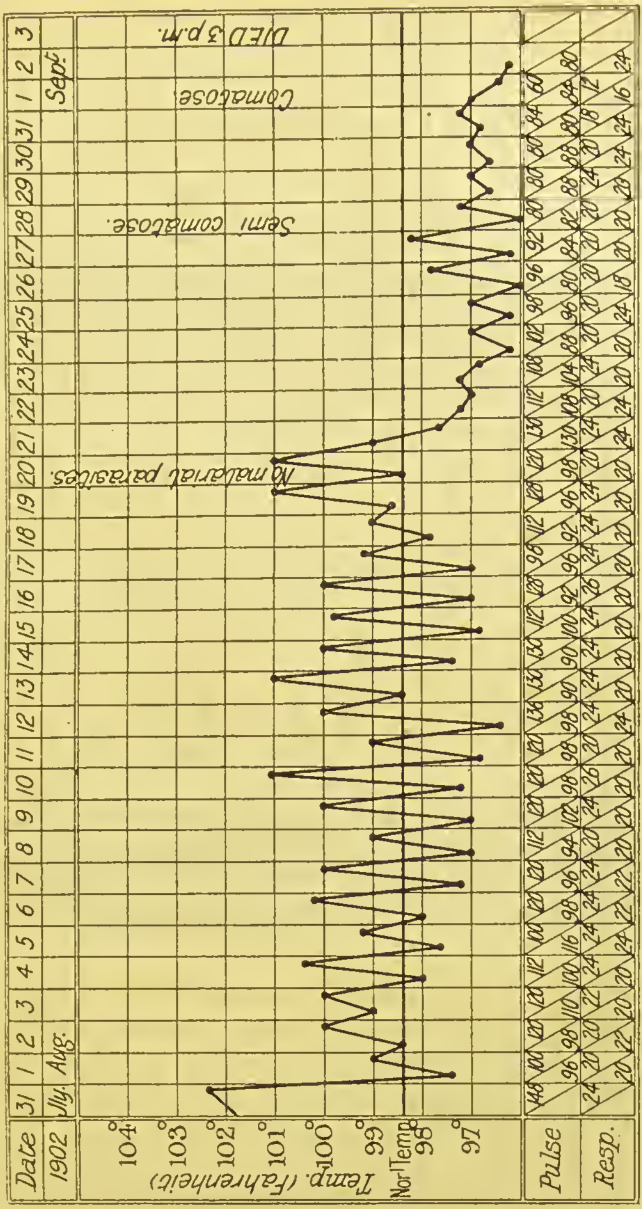


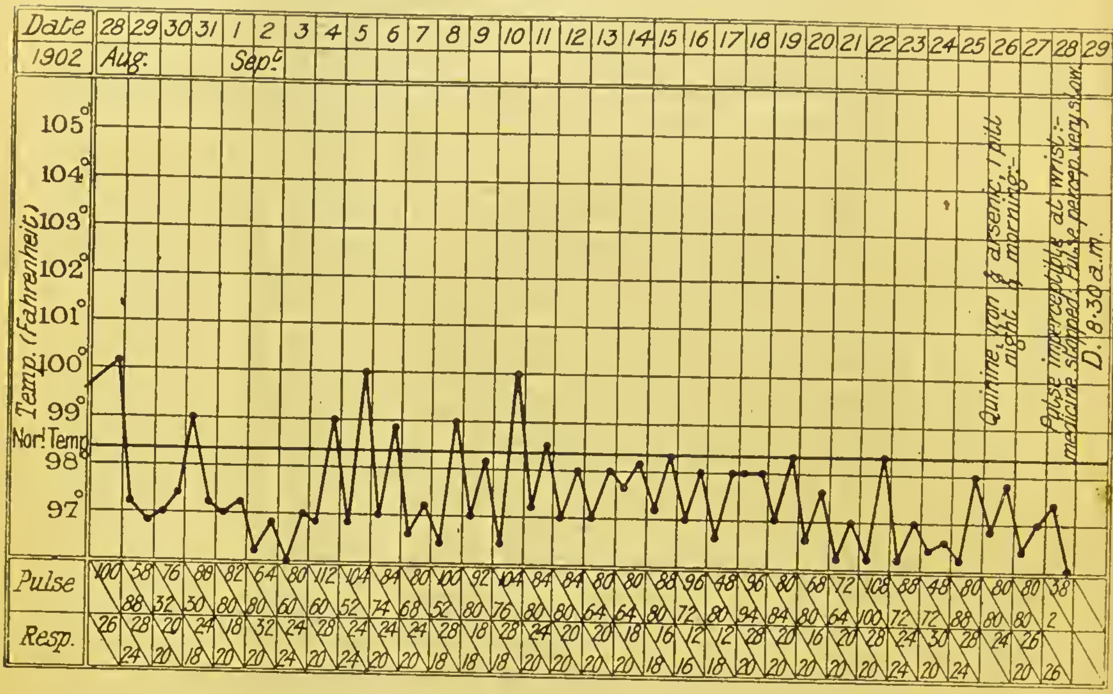
Chart 5.



This regular type of temperature may go on for three or four weeks, and then it may become distinctly irregular, sometimes almost keeping normal, with the exception of a slight evening rise now and again; this condition persists for a variable time, or it may again become regular. Just before death, one week or two weeks before, the temperature almost always becomes subnormal and remains so during the whole day. This latter condition warns one that the end is near. The rise of temperature is not accompanied by any special subjective symptoms, there is no rigor and no sweating, and it is not uncommon to see a patient with pyrexia of 103° F. walking about apparently with little the matter.

Variations from the typical temperature curves are from time to time met with, the chart below, with the exception of one or two evening rises, showing a practically subnormal temperature throughout the course of the disease; the case, however, was an acute one, and ended quickly in death.

Chart 6.—Atypical Temperature Chart.



In another type of case the temperature may remain high for several days without showing any marked morning remission. As a rule, however, after a few days it reverts to the usual type—no cases were ever seen by us without pyrexia at some part or other of the course of the disease. The ordinary temperature curve may be modified by intercurrent diseases, the most frequent being malarial fever; here the maximum is unusually high, often reaching 103° F. or over, and the remissions are slight, sometimes never coming below 100° F. Parasites may then be demonstrated in the blood, and the administration of quinine causes the temperature again to revert to its ordinary course.

### *The Circulatory System.*

The study of the pulse in Sleeping Sickness is very important, especially in the early diagnosis of the disease.

The frequency is very rarely below normal; it is usually of a rate of 90—130 per minute, but great variations may be got at different times of the same day. The ratio to the temperature is inconstant, a very high frequency often being associated with a low temperature, but in the last stages a low frequency associated with the subnormal temperature is the rule.

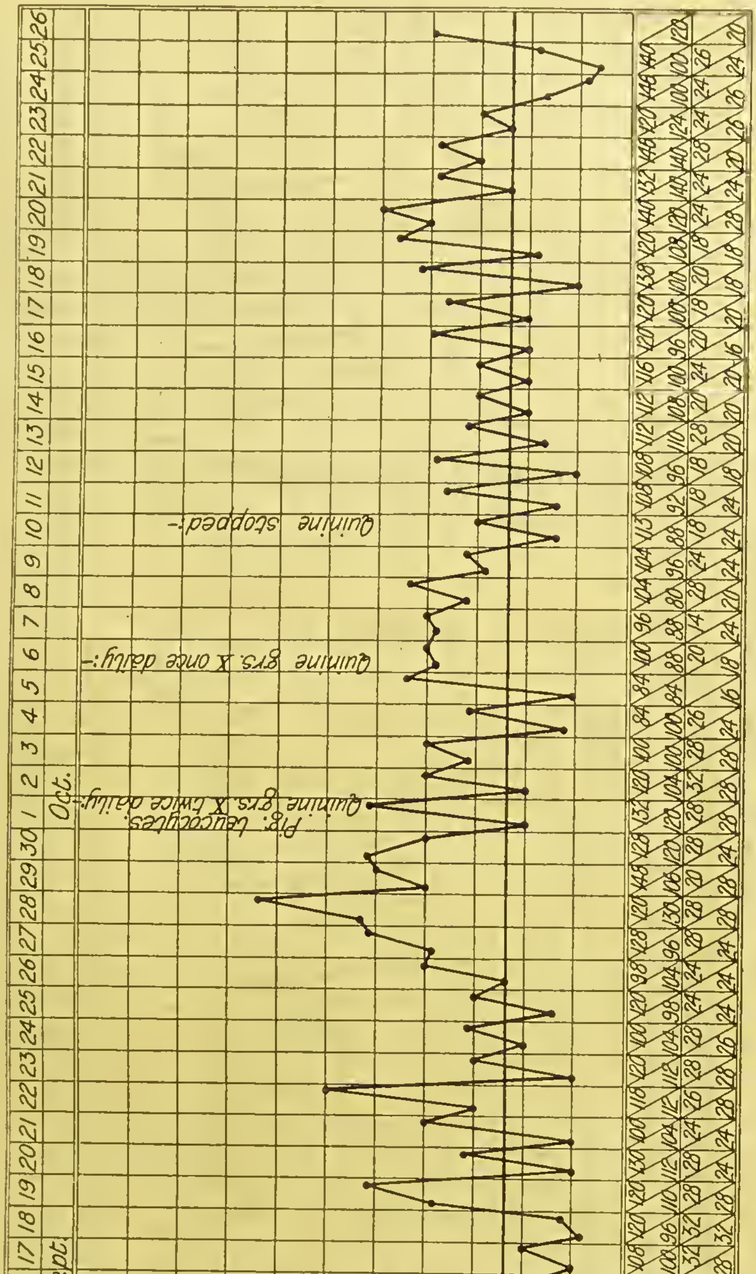


Chart 7.—Malarial Complication.

The rhythm is almost always regular in time and equal in force till the last stages, when some of the beats may be weaker than others; just before death, or even two days before, the pulse becomes imperceptible at the wrist. Dicrotism is uncommon, but it may be present when the fever is very high.

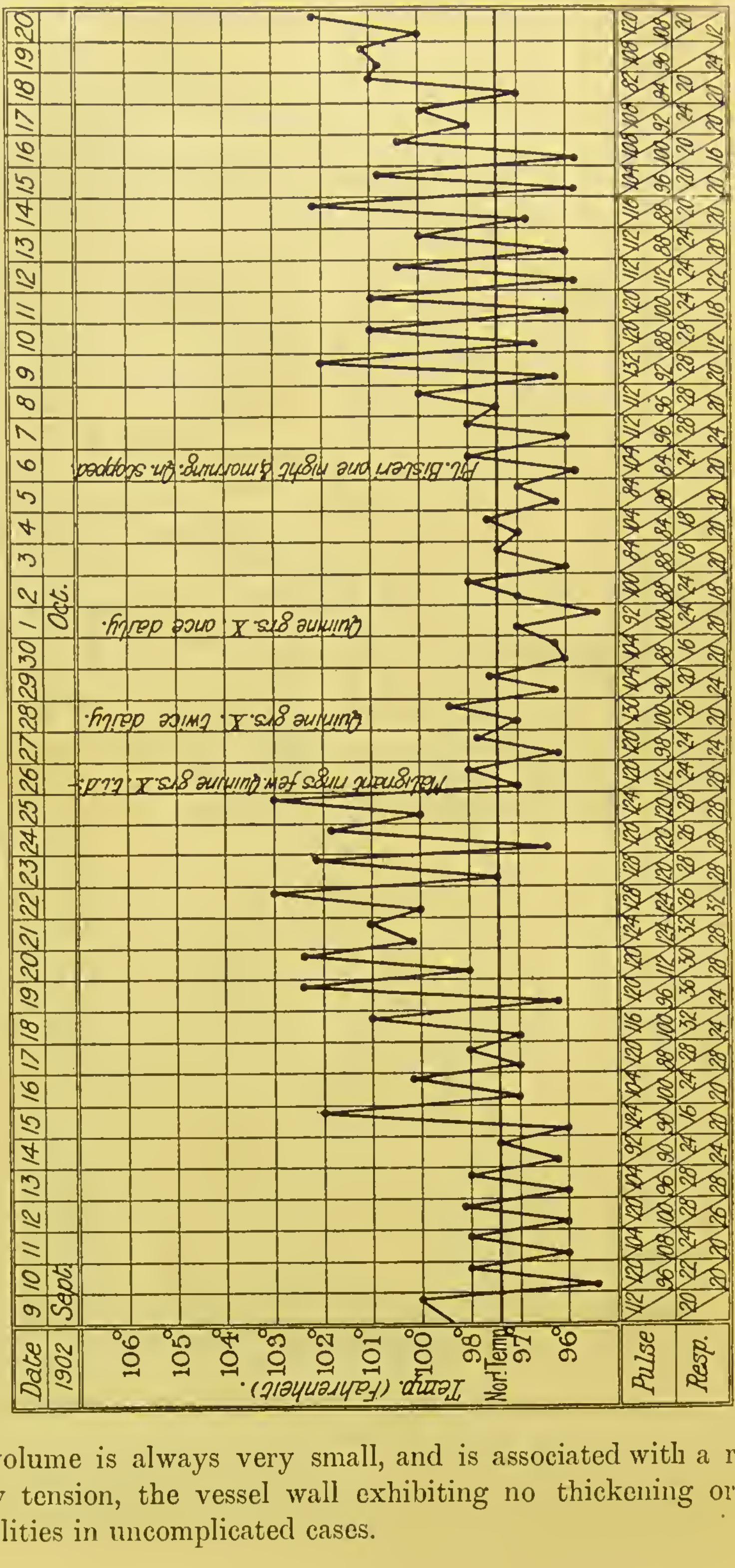


Chart 8.—Malarial Complication.

The volume is always very small, and is associated with a remarkably low tension, the vessel wall exhibiting no thickening or other abnormalities in uncomplicated cases.

There are no subjective symptoms as regards the heart; palpitation, cardiac pain, dyspnœa and giddiness are all absent; the physical examination shows little abnormal with the exception of the increased rhythm, and in some instances an increase in the transverse diameter. Inorganic bruits may be present, but neither valvular diseases nor endocarditis ever complicated any of the cases.

### *The Respiratory System.*

The respirations, regular in time and equal in force, are always increased, especially so in the evening; they correspond fairly closely to the increased pulse rate, and their number per minute generally is between 20 and 30. In the last stages of the disease Cheyne-Stokes breathing is common.

The physical examination of the respiratory system in the first stage reveals nothing. In the later stages some congestion and œdema of the bases of the lungs, due no doubt to the recumbent posture in bed, are commonly met with, and pneumonic patches may appear.

### *Alimentary System.*

In the early cases there is not much to be noticed in this system. In some patients the appetite appears to be slightly increased, and, even in the advanced cases, when completely bedridden, nourishment is still readily taken. Dyspeptic symptoms are as a rule absent, and vomiting is never seen. The bowels are constipated. Herpetic eruptions are never seen on the lips; ulcerative stomatitis in a slight degree is sometimes seen, and the gums in rare cases become swollen and spongy. The tongue is flabby and covered with a dirty fur; the papillæ on the dorsum may be prominent, and, in the last days of life, sordes tend to accumulate in the mouth. The breath often has an offensive odour. An inspection of the abdomen often reveals a certain degree of bulging; the stomach is generally confined to its normal limits, though a certain amount of dilatation may be present, due no doubt to the large amount of vegetable food eaten. The spleen is always enlarged, hard, and not tender, but the question of antecedent malaria in producing this must always be considered. The same may be said of the liver, though the degrees of enlargement of this organ are not so marked as those of the spleen. Pressure on the abdomen never elicits pain; the colon, especially in emaciated individuals, may often be felt full of hard fæces.

### *Integumentary System.*

Roughness of the skin, which has been considered so diagnostic of this disease, is not, according to our experience, by any means a constant feature, many cases exhibiting a perfectly smooth skin up to the day of their death. The same must be said of the eruptions, which are not frequent, and the type of eruption when existing is similar to that seen very commonly amongst the healthy natives. The most common eruption is a papulo-pustular one; the lesions are most frequently seen on the dorsum of the hands, extensor aspect of forearms and on the back. The pustules are generally isolated and have no tendency to run together. Pruritus often accompanies this condition, and the skin, as in other marasmic conditions, may, especially in the chronic cases, become rough, scaly, and lose its lustre. Forms of eczema and ordinary scabies are sometimes seen.

### *Lymphatic System.*

Enlarged glands in most descriptions of this disease have had special stress laid upon them. A special study of many natives, healthy or suffering from other ailments, shows at once that this condition is a common one, and cannot therefore be regarded as a special symptom of Sleeping Sickness. What produces this polyadenitis is difficult to determine, as it occurs in young children and also in adults. Perhaps frequent skin diseases, syphilis, and verminous invasions may be responsible. These enlarged glands are most commonly found in the posterior and anterior triangles of the neck, the submental region, the submaxillary region, Scarpa's triangles, and in the groins. In emaciated individuals the abdominal ones can also be felt. Their size varies from a small bean to a hazel-nut, and they are always hard and firm in consistence. They never become adherent or cause ulceration of the skin. The condition generally is a chronic one, though in some rare instances suppuration may be seen.

### *Nervous System.*

The dull apathetic look is one of the most characteristic features of the disease, though far from being a constant one. The expression is heavy, and shows very little emotion. There seems to be a slight loss of intelligence, but memory is not impaired. When spoken to they respond after a considerable interval. Speech is not, however, specially affected, there being no stammering nor slurring in the production of the individual syllables.

Proper sleep is not a symptom of the disease as is so generally supposed; a patient if left alone is often seen to nod his head and close his eyes, but the slightest touch, such as stroking him, or any noise, will make him open his eyes at once. The total amount of sleep may be above the average in a few cases, but the usual feature is one of lethargy, indifference, and drowsiness, and even this in several cases is not well marked, while in others it may be entirely absent.

Maniacal attacks sometimes usher in the onset of the disease. Headache, chiefly occipital, indefinite pains in the chest and sometimes in the joints, especially in the knees and ankles, are sometimes complained of.

*Sensory Functions.*—These at first are generally normal, though even early there may be some hyperæsthesia at the trigeminal points. Touching or moving the patient, especially in those cases where flexure contraction or rigidity of the muscles of the neck is marked, causes pain, evidenced by the patient crying out. General or local anæsthesias were never observed. The temperature sense is unimpaired, and the same seems to be the case with the muscular sense, though this is difficult to be sure of in dealing with savages.

*Motor Functions.*—Especially towards the end of the illness, when emaciation may be marked, the muscles become frequently wasted and flabby, though in the acute cases death often happens without much loss of the muscular nutrition. The motor-power, at first powerful, diminishes towards the end. In many cases a certain degree of incoordination is distinct, and in a few cases Romberg's symptom is present. "The gait is a typical one and best described by the term 'shuffling,'" the feet not being raised from the ground, but being pushed forward.

Abnormal muscular movements are perhaps the most striking feature of the disease; fine tremor in the tongue is very constant, and a somewhat coarser tremor is also usual in the hands and arms, any purposeful movements, as lifting a cup to the lips, often increasing it. In a few instances tremor is found in the legs and muscles of the trunk so excessive as to cause shaking of the bed in which the patient lies. In a few rare cases, which were ultimately proved at the necropsy to be undoubtedly Sleeping Sickness, no tremors were visible during the whole course of the disease.

In the last stages rigidity is common in the muscles of the neck and flexure contractions of the legs on thighs, and the thighs on the abdomen may at this time be extreme. Fits of an epileptic nature sometimes occur, either general or localised to a group of muscles.

Paralysis is rare; it was once noticed in the muscles of the right side of the face, and in another case in one arm. Hemiplegia or paraplegia were never seen, and choreic movements were never observed.

*Reflex Functions.*—The superficial reflexes are generally normal. The deep, exaggerated at first, afterwards become lost. There is never clonus. The organic reflexes are normal, but during the last weeks the motions are passed involuntarily, this, no doubt, depending on weakness and inability to get out of bed rather than on any definite paralysis.

Babinsky's symptom is never present.

Lumbar puncture was performed in the great majority of cases.

The cerebro-spinal fluid escapes generally with increased pressure, though in a few cases the pressure may be normal or even decreased. The fluid appears as a rule perfectly clear and colourless. In some few cases it is slightly turbid. Albumen is found only in traces. The liquid always reduced Fehling's solution. The leucocytic formula is mononuclear.

*Special Senses: Eyes.*—The pupils are equal and as a rule dilated. They contract to light and during accommodation, though in some cases both reactions are sluggish. There is never nystagmus nor any ocular paralysis. (Hearing, taste, and smell are, apparently, unimpaired.) There is no change in the fundus or retina.

### *Urinary System.*

As has been mentioned before, weakness of the sphincter vesicæ has been frequently met with in the last stages of the disease. We have never observed retention, nor any temporary difficulty in starting micturition. The amount of urine passed during the 24 hours is generally normal at first, while in advanced cases a distinct increase of the secretion may be noticed. The urine when freshly passed is generally clear, and of a very pale colour. It is of a low specific gravity and does not give any characteristic odour. The reaction is almost constantly alkaline, this probably being due to the vegetarian diet of the patients. Carbonates are abundant and also earthy phosphates. The amount of urea excreted is below the average. Albumen is absent as a rule, though a slight trace of it may be present sometimes, when the pyrexia reaches a high degree. Sugar is always absent, and so are bile pigments in uncomplicated cases. The sediment is chiefly composed of oxalates, triple phosphates, and carbonates.

### *Sexual System.*

At first sexual desire is apparently not decreased, but later it is lost as the sexual system partakes of the general nervous debility. Menstruation may remain normal for a long time, but disappears as a rule in advanced cases. In the mucous secretion of the vagina, which is frequently increased and of acid reaction, one often finds in several cases a form of flagellatum with three flagella and an undulating membrane, showing very active movements (Trichomonas vaginalis) (Dormé). The same flagellation was observed also in the vaginal secretion of apparently healthy women.

### *Fæces.*

Constipation, as already mentioned, is very frequent. The stools are generally hard, of greenish colour, and almost free from odour. Portions of undigested food, large shreds of vegetable tissues, and long fibres are often to be seen. This appearance of the fæces depends certainly on the diet of the patients, which consist practically of vegetables only. Microscopically there are innumerable micro-organisms, epithelial cells, crystals of triple phosphate, oxalate of lime, and occasionally Charcot's crystals; there are also many vegetable cells, starch granules, and frequently yeast fungi. Ova of different parasites are always present. The commonest of these are Ankylostoma duodenale and Ascaris lumbricoides; less common, Trichocephalus dispar. In two cases Anguillula intestinale was observed. In several instances ova of Bilharzia hæmatobia were present in the fæces. The ova had the characteristic spine, not at one pole but a little to one side. Some had no spine at all; free embryos were also observed. Among the protozoa Trichomonas intestinalis is very common.

## *Hæmopoietic System.*

Anæmia in varying amount is constant: the average number of the red blood corpuscles per c.mm. being 3,500,000 or thereabout. So many other blood-destroying factors are present, such as malaria and ankylostomiasis, that it is difficult to say what part they also play in producing this condition of the blood. In some cases if cyanosis exists just before death there may be an abnormal increase in the R.B.C., one such case showing 6,200,000 per c.mm. on the day of death. Commoner than this, however, is a gradual fall to 2,000,000 or under. The hæmoglobin is generally reduced in relationship to the amount of the anæmia. The leucocytic count shows no absolute increase from the normal until just before death, when a certain number of the cases get a well-marked terminal polymorphonuclear leucocytosis. Relatively the large mononuclear elements are increased; but malaria must again be taken into account in considering this. Especially in young subjects a relative increase in the number of eosinophile leucocytes is often met with; this is probaby due to helminthiasis.

The main features of the blood may be summed up as follows: An anæmia of varying degrees, with a relative increase of the large mononuclear leucocytes, malarial parasites and pigmented leucocytes, indicating malarial complications, are frequently met with.

## *Complications.*

Bed sores, when emaciation is extreme, are common, especially so when one considers the nature of the beds in which the patients are treated. With ordinary care they may be largely avoided. They are most apt to be got in the chronic type of cases. As already stated, under the integumentary system skin affections of varying nature are common. In a few cases boils of a pemphigoid nature were observed, the distribution of these being limited to a few spots. Ulcerations of the hands and feet due to chiggers are common.

Epistaxis is rare. Different forms of laryngitis were met with, the cause of death in one case being œdema of the glottis. Bronchitis, œdema, and nasal congestion are all common in the last stages. Lobar pneumonia is one of the most frequent causes of death. In children and old people broncho-pneumonia is common.

Valvular diseases of the heart and affections of the kidneys are extremely rare.

Sometimes pseudo-dysenteric symptoms, due to the presence of bilharzia parasites, were observed.

### *Course of the Disease.*

The incubation period is an uncertain one, and there is the greatest difficulty in determining this point, because of the very insidious onset of the disease. The rapid spread of the disorder in Kavirondo would seem to show that the incubation period may be a more rapid one than is generally supposed. After the stage of incubation is over the symptoms, as stated in the general sketch, begin very insidiously, and the course of the disease is a very variable one. The average duration is from 4 to 8 months, and not infrequently cases much more acute than these are seen. Very chronic cases, running a course of more than a year's duration, are very rare.

The disease clinically may be divided into three stages. The first stage is represented by somewhat indefinite symptoms, vague pains, feelings of indisposition, and sometimes a slight degree of drowsiness. Fever is always present (an important point). The second stage may be designated as the stage of tremor. With an increase of the former symptoms, tremors accompanied by other nervous symptoms appear. The third stage is characterised by intense weakness. The patients now become bedridden, they loose control of their sphincters, a coma which progressively deepens appears, temperature falls to subnormal, and the patient passes away.

### *Pathological Anatomy.*

The pathological changes as pointed out by Mott are essentially those of a chronic meningo-encephalitis, and a meningo-myelitis. Macroscopically in the large majority of the cases there is not much to be seen. The calvarium is not thickened. The dura-mater shows little change; it is seldom adherent to the skull, but may be to the subjacent arachnoid. The sub-dural fluid is not usually in excess, though some cases show a marked increase. The pia-arachnoid, generally clear, is, however, sometimes opaque and slightly thickened. In some instances it is adherent to the brain, and on being stripped off causes some erosion;

the vessels in this membrane may show some congestion. The subarachnoid fluid is usually pale straw coloured and is often in excess. In a few cases it is slighty turbid, and in two instances was distinctly purulent, especially over the sulci and round the vessels in the membranes. Recent lymph in small amount may be found. On section the substance of the brain is moderately firm and nothing abnormal is to be seen, with the exception sometimes of excess of fluid in, and dilatation of, the lateral ventricles. The pituitary body, the pons cerebellum and medulla oblongata show no macroscopical changes. The membranes covering the spinal cord show the same appearance as that of the brain, and sections of different areas reveal nothing abnormal macroscopically.

The other organs also show little change. The heart is usually flabby, pale, and may show signs of myocardial changes. Congestion and œdema of the lungs are almost always seen. Patches of bronchial pneumonia are common. Pleural effusion was never observed, though in some cases pleuritic adhesions were noted.

The livers in all the series of cases examined showed distinctly the appearance of chronic malaria, and the same was also found in the spleen, which was often considerably enlarged. Section of the spleen showed the characteristic pigmentation of malaria. The kidneys, suprarenal capsules, and pancreas were in all cases normal. The stomach, usually of an ordinary size, may sometimes be dilated, and may show a patchy congestion, the intestines often exhibiting a similar congestion. Parasitic nematodes, *Ankylostoma duodenale*, and *Ascaris lumbricoides*, were invariably present. The large intestines in four cases exhibited marked congestion and superficial erosions of the mucosa due to the ova of Bilharzia hæmatobia. *Trichocephalus dispar* was always found in the cæcum. In the mesentery the adult forms of *Filaria perstans* were often found in the connective tissues, and bilharzia adults in the mesenteric veins.

Enlarged glands in all cases were a general feature; in the superficial areas the size varied from a bean to an almond, but the retro-peritoneal ones were often much larger, sometimes reaching the size of a walnut. This condition, however, was met with also at the autopsy of cases suffering from other diseases. The bladder, urinary and sexual organs were never affected.

Microscopically definite and characteristic changes are always to be met with in the nervous system. A mononuclear leucocytic infiltration is always seen on the meningeal surface of the brain spreading down into the sulci, and affecting in varying degrees the perivascular spaces around the blood vessels in the substance of the cerebrum pons, medulla and spinal cord. The leucocytic formula of the cerebro-spinal fluid is also mononuclear.

The degree of perivascular infiltration varies considerably and does not follow a corresponding ratio to the symptoms. The nerve cells show frequent pathological changes, being often altered and irregular, very much resembling those described by Mott. The tangential and inter-radial association fibres are diminished in number, and many show great atrophy. Sections stained by Loeffler's and Gram's methods never showed micro-organisms, with the exception of four cases in which cocci which retained the Gram's stain were seen in couples and short chains.

The pia-arachoid of the spinal cord showed a similar leucocytic infiltration, which was also seen in varying degrees around the vessels in the substance of the cord. The axis cylinders and medullary sheaths of the nerves in the cord (stained by Marchi's method) may show a diffuse degeneration in some cases.

The arteries both in the brain and spinal cord never exhibited signs of endarteritis. The peripheral nerves (sciatic, anterior and posterior, tibial, ulnar and radial, etc.) showed no signs of degeneration, with the exception of deposits of fat in the epineurium and sometimes in the endoneurium. As regards the other organs of the body there is not much to note: a leucocytic infiltration may be seen in almost all the organs, and especially in the heart, and secondary changes such as occur in infectious diseases may also be got, namely, cloudy swelling of the liver cells and degenerative changes in the myocardium.

The liver and spleen of all the cases examined in the *post-mortem* room in Entebbe showed chronic malarial changes, and plenty of recent or old malarial pigment. Sections of the spleen stained for micro-organisms showed in very few cases the same cocci as described in the brain. Sections of the bilharzial erosions showed a marked hæmorrhagic condition of the mucous membrane, and the presence of many typical ova. No infections of the bladder by bilharzia were seen, and it would thus appear that rectal bilharzial disease is the common form in Uganda, the ova in all such cases being furnished with a lateral spine.

## *Diagnosis.*

The diagnosis in early cases may be exceedingly difficult, as the typical features of the disease (tremor, etc.) are generally absent. The most important fact on which to base the diagnosis in this stage is the evening rise of temperature and the increased rate of the pulse. Later on, when the definite symptoms appear, there is little difficulty, as the disease does not resemble closely any of the common nervous disorders, with, perhaps, according to Mauther, the exception of Wernicke's acute poliencephalitis superior.

## *Beri-Beri.*

Sleeping Sickness has been considered by some authors to be a form of Beri-Beri. To anyone with experience of the two diseases such a mistake should never occur. The latter disease is a peripheral neuritis which comes on rapidly. In the wet form there is marked œdema, in the dry form, wasting of the muscles. In both forms the knee-reflex is abolished, and hyperæsthesia of the muscles is a prominent feature. In Sleeping Sickness these symptoms are absent, and the tremor, the pyrexia and the lethargy at once distinguish the two diseases.

*Intracranial Syphilis and Tumours.*—Some cases of intracranial syphilis and tumours of the brain have been described, with a curious tendency to somnolence, so that the person drops off to sleep while at work. Such cases if occurring in the endemic area might present difficulties, but in the former other evidences of syphilis would be present, and in the latter the usual definite symptoms of a cerebral tumour would be found. The temperature which is such a constant symptom of Sleeping Sickness would greatly clear the diagnosis.

*Chronic Nephritis.*—Chronic nephritis with uræmic symptoms may present some superficial resemblance to Sleeping Sickness. Such a case was sent to our hospital in Entebbe, with the diagnosis of Sleeping Sickness, but a careful examination of the case revealed the fact that the patient's urine contained albumin, and that there was also a well marked albuminuric retinitis. At the autopsy a few weeks afterwards the pathological examination upheld the diagnosis.

*Tabes and General Paralysis of the Insane.*—In some rare cases the pupils may react to light very sluggishly, and if Romberg's symptom and the absence of the knee jerk also be present, the question of tabes dorsalis must be considered. Strangely enough, though syphilis is prevalent in Uganda, parasyphilitic diseases, according to Cook and to our own experience, are excessively rare. Tremor and pyrexia will lead one to the diagnosis of Sleeping Sickness. Perhaps the disease which most closely resembles Sleeping Sickness is general paralysis of the insane, as in the former disease symptoms of insanity are not infrequently seen. The regular pyrexia almost constantly present in Sleeping Sickness is the best test to differentiate the two diseases.

## *Prognosis.*

The prognosis is a grave one, as the disease always terminates fatally. All the cases in our hospital in Entebbe died, and we were never able to satisfy ourselves as to rumours of people recovering. A native doctor claimed to have cured several patients by incisions over the temples, but the French Missionaries told us that all those patients, after a short apparent improvement, died. The disease appears to be invariably fatal.

## *Treatment.*

Many drugs were tried without any definite results. Iron, arsenic, and quinine, especially in the cases complicated with malaria, produced a distinct but temporary improvement. Free opening of the bowels with magnesium sulphate or castor oil to counteract the persistent constipation gave considerable relief. The thermocautery and blistering the head and spine with iodine always caused a distinct temporary alleviation of the symptoms.* Otherwise drugs did not appear to do any good. With care, cleanliness, good feeding and proper attention, life may be prolonged for a considerable period, but the end comes sooner or later.

## CLINICAL RECORDS OF A SERIES OF CASES OF SLEEPING SICKNESS.

CASE 1.—Gerako. F. 20. Ad. July 15, 1902. Died July 31, 1902. Native of Buse, Uganda :—

*Family History.*—Impossible to get. One brother said to come to visit her, apparently healthy. No other cases of Sleeping Sickness said to be in family. Other points not obtainable.

*State on Admission.*—Found lying on ground outside a native hut in a state of semi-coma; marked tremor of tongue and other muscles. On admission to hospital, lies in bed, takes no interest in anything. Lies more in a state of semi-coma than sleep. Eats a little when forced. Speech practically nil, apparently does not understand. Passes motions in bed.

*Circ. System.*—P. 120, reg. equal. Vol. small, tens. low. V. W. unthickened. H., u. b., 3rd rib, l. b. 3½ inches from m. s. l., r. b. ¼ inch from m. s. l., A.B. 5th sp., 3 inches from m. l., no visible puls. Aus. Sounds feeble, no murmurs.

*Resp. Syst.*—R. 22, reg. shallow, thor. abdominal.

Palp., V.F., not impaired. Per note res. both sides all over. Aus. vesic, no prol. expir.

*Aliment. Syst.*—Bowels constip. Palp. nil, muscles somewhat rigid. Perc. Liver, u. b., r. d., 5th rib, a. d., 6th rib, l. b. c., mar. spleen, u. b., 8th r., l. b. c. m.

*Stomach* not enlarged.

*Nerv. Syst.*—Semi-unconscious, not drowsy or sleeping. Tremor very marked in tongue, none in face, seen in muscles of hands and arms; both sides very marked, also legs on both sides; none abdom. or thorax muscles; now and then a coarse twitching in muscles of forearms both sides.

Motor functions. Reflexes. Sup. present, marked plantar, and epigastric deep, knee present, diminished, more marked left than right. No ankle clonus.

*Sensory Functions.*—Pain, touch, and temp. sense present, not increased, ordinary. Motions passed in bed. Legs weak, not wasted in appearance, no def. paralysis, grip feeble, loss of motor power.

*Special Senses.*—Pupils equal, react. to light and during accommodation. Smell and hearing unobtainable. No paralysis eye muscles.

*Urinary System.*—Examination. No albumin, blood or sugar.

*Lymphatic System.*—Slight glandular enlargement under jaw on both sides, small, also slight in groins, more marked left than right; other situations, none palpable.

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* The injection of a solution of sublimate into the veins, as recommended by Baccelli for other diseases, caused a fall in the temperature, and this condition persisted for several days.

*Further Notes*—July 26, 1902. Not much change, tremors a little more marked. Does not sleep much, but is very dull and apathetic, mumbles only in reply to questions.

July 30. Worse. Feeds very badly, and, on swallowing milk, has some difficulty; pain in neck on pressure, and apparently pain on swallowing. Marked swelling has now appeared in sub-mental region of right side, tense, and exceedingly tender; extends over mesial line to left. Tremors about the same in extent, no def. signs of paralysis. Motions passed in bed. Temp. not up. Attempt to examine throat impossible, resists strongly, and has apparent difficulty in opening mouth. No physical signs in lungs.

July 31. Died suddenly about 4 A.M.

*P.M.* 8.30 A.M.; 4½ *Hours after Death.*

*External Appearances.*—Skin soft to touch. No scratch marks or slightest signs of eruptions. Body well nourished, no emaciation, no wounds or old scars, no bone thickening, large swelling, elastic in consistence, on left side under jaw, extending up into parotid region and slightly over mesial line; glands palpable on both sides of neck, also in rg. axilla, slight in left, also in groin.

*Section.*—Well marked layers of fat sub-cutaneously over thorax and abdomen.

*Thorax.*—Retrosternal glands enlarged.

*Pericardium.*—Small amount of clear fluid in sac.

*Heart.*—Not enlarged. Valves competent. No alterations in muscles. No atheroma aorta.

*Lungs.*—No pleurisy or pleuritic fluid. L. L. A little cong. base. No pneumonia. R. L. No pneumonia. Congestion lower lobe.

*Abdomen.*—No peritoneal fluid. *Liver* slightly enlarged. Normal in consistence. *Gall Bladder.* A little greenish bile.

*Spleen.*—Enlarged slightly. Slate coloured capsule. Section dark, pigmented.

*Kidneys.*—Capsule strips easily in both. Section nil.

*Pancreas.*—Nil.

*Intestines.*—Small, nil; large, full of fæces. Appendix normal. Ascaris lumbricoides many. Ankylostomes, many in duodenum. Trichocephalus dispar in colon. All mesenteric glands greatly enlarged, matted together in places, some as big as walnuts, hard in consistence, no pus.

*Mature F. perstans.*—One entire and one portion of an adult in tissue of mesentery near root.

*Bladder.*—Empty, no urine.

*Uterus and Vagina.*—Nil.

*Ovaries.*—Both enlarged. L. three times normal size, R. about twice.

*Neck.*—Large swelling noted in life, due to a mass of glands under jaw of all sizes. Sub-mental necrotic breaking down. Soft in consistence. All glands in both triangles enlarged. A large gland, size of a big walnut, in post triangle of rg. side, pus in its interior.

*Tongue* healthy. Tonsils enlarged, follicular tonsillitis. *Larynx*, extreme œdema glotidis, tissues also around œdematous. Thyroid apparently healthy. *Pharynx*, pharyngitis.

*Brain.*—Dura adherent at places, a considerable amount of clear fluid. Vessels over surface very congested. Pia arachnoid markedly adherent. A large amount of distinctly turbid fluid, especially over the sulci and round the vessels under the membrane. Picture of meningitis not so marked on base of brain.

*Section.*—Tissue of brain firm. Both lateral ventricles distended and full of fluid, nuclei and thalamus nil. Pons and medulla and cerebellum nil macroscopically. Cord not examined.

*Lymphatic System.*—General enlargement of glands all over body. Special regions already noted.

*Histological Examination.*—Old malarial pigment in spleen and liver. No malarial parasites in spleen. Smears (measured) of blood from different organs and vessels. No def. results. Smears from glands, no Filariæ, pus and all glands full of diplococci. Sub-dural and sub-arachnoid fluid, no Filariæ. Pus cells and diplococci.

*Brain.*—Picture of a meningo-encephalitis. Pia arachnoid, well marked inflammation, considerable leucocytic exudation. Vessels in interior, with their peri-vascular lymphatics, packed with mononuclear leucocytes. Diplococci numerous, but apparently Sleeping Sickness changes first, then complication of micrococcic infection.

*Pituitary Body.*—Section thro. ant. portion, capsule thickened, some exudation. Interior nil.

*Thyroid.*—No decrease of colloid material.

*Ovary.*—Fibroid tissue increased. Hæmorrhagic areas in places.

*Tonsil.*—Erosion of epithel, covering at places, round-celled infiltration beneath this vessel congested. Increase of monuclear leucocytes.

*Glands.*—Bronchial, two. One shows very little, also other.

*Neck.*—One shows intense congestion of blood-vessels, and dilation of those lymphoid tissue increased.

No. 2. More marked than first. Enormous dilation of capillaries engorged with blood.

No. 3. Not much change. No dilated capillaries, increase of lymphoid tissue.

Case 2.—Caperi. M. 8. Ad. July 15, 1902. Dis. Native of Entebbe, Uganda :—

*Family History.*—Impossible to find out. F. 1 said to have S. S., doubtful. Duration of illness said to be two months.

*Pers. History.*—Found at Busi, when seen very apathetic and drowsy, nodding his head when sitting; walking impaired, pulls feet along after him resembling locomotor gait. Taken to Entebbe.

*State on Admission.*—No emaciation, eats, understands when spoken to and obeys directions. Drowsiness marked, but does not really sleep. No tremors visible, glands enlarged. F. P. in blood.

*Circ. Syst.*—P. 80, reg. equal. Vol. very small, tens. low. V. W. nil, no visible puls. a. b. 5th space u. b. 3rd rib, l. b. mam. line, r. b. ¼-inch, rg. sternal border, aus. sounds faint, no murmurs.

*Resp. System.*—R. 18 equal, thor. abdomen, no cough. Note res. all over. Sounds vesic. somewhat faint.

*Alim. System.*—Insp. nil, pal. sp. and liv. palp. Liver u. b., 5th r. a. d., 6th l. b. 1 inch below c. m. Spleen u. b., 7th rib l. b. 1 inch below c. m. Bowels constip.

*Nerv. Syst.*—Drowsiness, no tremor in tongue or arms. *Mot. F. K.* reflexes pres. ordinary plantar cremasteric, epigastric well marked. No wasting or paralysis of muscles. Walks peculiarly, dragging one leg after other. Muscular power? Ankle clonus nil.

*Sens.*—Touch pain and temp. sens. present, legs, arms, abdomen.

*Spec. Sens.*—Eyes, pupils equal mod. dil., react to light and accom., no nystagmus, no ocular paralysis. Hearing apparently unimpaired.

*Urinary System.*—No subject. symptoms. Quant.? sp. gr. 1020, sl. alkaline. No alb., bl. or sugar. Deposit nil.

*Lymphatic Syst.*—Glands palpable, ant. post. neck both sides and all other situations, e. g. gen. enlargement.

*Further Notes.*—July 26. Temp. fluctuating, irreg., pyrexia; condition generally unchanged, retains command over sphincters, passing motions ordinarily.

July 30. Pyrexia to-night, nothing else, eats well and is less drowsy.

August 1. Seems much better, up during day. Walks better and does not drag his limbs so much. Eats well. No paralysis of sphincters. Tendency to drowsiness. No tremors.

August 2. No change except the detection of a very fine tremor now and then on dorsum of tongue.

August 5. Very fine tremor in tongue at times. Knee reflexes present. Now diminished distinctly. Marked tendency to drowsiness to-day. Pupils equal, mod. dilated. Glands, no increase in size since July 15. Sp. punc. 5 P.M.

August 13. No further change. Quinine 5 grs., mag. sulph. ʒ j. twice daily.

August 19. Better, runs about out doors all day, no marked tendency to sleep.

August 28. Great rise in temp. (102). Drowsiness much more marked. General conditions worse.

August 29 and 30. No change.

September 1. Better, not so drowsy.

September 3. No change.

September 8. No change.

September 11. Rather brighter, no tremor tongue or hands, still drowsy look.

September 24. *In statu quo.* Runs about all day. Still drowsy.

September 29. *In statu quo.*

October 5. *In statu quo.*

October 10. No change. Brighter. Stays about doors during day. Not much drowsiness.

October 28. Has remained in same condition since 10th. Still sometimes a heavy dull look, not markedly stupid, however.

## Table of Blood Counts.

| 1902. | | |
|---|---|---|
| July | 24. | R., 3,900,000; L., 6200. |
| August | 6. | R., 3,560,000; W., 8400; Hb., 78 per cent.; P. M. N., 39 per cent.; L. M., 30 per cent.; l., 16 per cent.; E., 15 per cent.; T., 0 per cent. |
| August | 14. | R., 3,500,000; W., 9000; Hb., 81 per cent. |
| August | 19. | R., 3,580,000; W., 7000; Hb., 78 per cent. |
| September | 10. | R., 3,780,000; W., 8700; Hb., 76 per cent. |
| September | 17. | R., 4,440,000; W., 6560; Hb., 80 per cent.; P. M. N., 32 per cent.; L. M., 6 per cent.; l., 39 per cent.; E., 20 per cent.; T., 3 per cent. |
| October | 28. | R., 4,400,000; W., 5000; Hb., 80 per cent. |

CASE 3.—Waniha. F. 25. Ad. July 15, 1902. Dis. August 28, 1902. Died. *P.M.* Native of Manyamgo, Uganda:—

*Family History.*—F.? M.? S. l. h. B. O. Ch. l. h.

*State on Ad.*—Duration of illness not known. Carried in from out district. Very feeble and weak, walking with a very marked shuffling gait. Tremor of tongue marked, also slight in hands. Glands in neck, etc. Pyrexia. No marked sleepiness, but somewhat drowsy and very apathetic. Ad. as an early case, not much wasting.

*Circ. Syst.*—P. 120. Reg. equal. Vol. small. Tens. practically nil, V.W. unthickened. A. B. not. palp. V. B. 3rd rib, l. b. 3½ inches from m.s. l. r. b. ¼-inch to rg. of m. s. l. Ausc. sounds faint at apex, better heard at base. No murmurs.

*Resp. Syst.*—R. 24. Reg. equal, thorax, abdomen. Condition of chest healthy, no dullness anywhere, breath sounds vesicular.

*Aliment. Syst.*—Appetite good, eats native food. Mouth, nil. Bowels generally constipated. Liver u. b. R. d., 6th a. d., 7th l. e. c. m. Spleen u. b., 7th l. b. cont. margin. Nothing else in abdomen.

*Nervous System.*—Intellectual functions: Apathetic, not evincing interest in anything except food. Does not sleep much, though at times apparently drowsy. No delirium or coma. Speech slow and unintelligent.

*Cranial Nerves.*—Smell impossible to obtain. Sight apparently unimpaired; recognises people. No paralysis eye muscles. Nystagmus nil. Pupils equal, mod. dilated. Reacts to light and accommodation. Fifth nerve unimpaired, no paralysis of face muscles, e. g. facial, other nerves not paralysed. No deviation of tongue.

*Motor Functions.*—No def. evidence of paralysis. Grip feeble and musc. power of limbs also feeble. Co-ordination impaired. Romberg's symptom very slight. Nutrition of muscles somewhat wasted and flabby. Fine tremor on tongue and in upper limbs.

*Sensory Functions.*—Ordinary senses well marked. Pain sense increased somewhat, temperature sense undiminished. Musc. sense unobtainable.

*Reflex Functions.*—Plantar abdom. Conjunctival present well marked. Knee reflexes increased a little on left side, about ordinary right, no ankle clonus or knee clonus. Organic reflexes not impaired.

*Gait.*—Peculiar, slow and shuffling.

*Urinary System.*—No subj. symptoms, sp. gr. 1029, no albumin Bl. or sugar. Dep. mucus. Quant?

*Lymphatic System.*—Glands palpable in neck, groins slightly, and in axillæ.

*Skin.*—No eruptions or any def. pruritus.

*Further Notes.*—July 22. Very bad. P. 130, almost imperceptible, very weak.

July 25. Somewhat better. Does not pass motions in bed.

August 1. Appears better. Gets up to verandah for food. Walk peculiar, does not raise feet far from ground but pushes them more, tends to stagger a little and has to support herself to get into bed.

August 2. No change.

August 5. Reflexes. Slight increase left as compared with rg., which is about normal. Very apathetic and stupid, not drowsy and no tendency to sleep.

August 13. Quinine 5 grs. Mag. sulph. ʒ ij. twice daily. No further change.

August 14. Diarrhœa since yesterday. Medicine stopped. Emaciation is now becoming marked. Very stupid, heavy expression of face. Rigidity of neck muscles appeared. Painful when touched, tremors increasing. Has remained in bed for four days, *e.g.*, since morning of 10th.

August 16. Emaciating rapidly. Passing motions in bed. Rigidity of neck, commencing contractions of legs. Pulse very feeble, no tension. Very ill.

August 18. Slightly better. Emaciation extreme. Slight pruritus to-day. No eruptions, tremors about the same. Still passes motions in bed.

August 19. Slightly better, took milk this morning readily. Passing motions in bed.

August 24. Extreme weakness. Very low temp. (96 F.). Steady pulse, tremor decreased, knee jerk cannot be elicited. The patient is constipated.

August 26. Several convulsive attacks. The head is pushed backwards. The muscles of the neck are contracted (during the attack only), twitching of facial muscles, more marked on the right side; the eyeballs look to the right, the pupils do not react to light, stertorous respiration. Attack lasts about 2-3 minutes and is soon followed by another.

August 27. Several convulsive attacks like yesterday.

August 28. In the morning a few convulsive attacks. In the afternoon well-marked Cheyne-Stokes breathing, the pulse cannot be felt any more at the wrist. Tremor very much decreased. The patient dies at 6.30 P.M. without showing any more convulsions.

## Table of Blood Counts.

1902.
August 6. R., 3,650,000 ; W., 7500 ; Hb., 75 per cent.
August 14. R., 3,500,000 ; W., 8000 ; Hb., 78 per cent.

*P.M.* August 29, 15 hours after death (body well preserved, as the night was very cool).

*External Appearances.*—Extreme emaciation, a superficial and not very large bed sore on right gluteal region, no skin eruptions. Glands palpable on both sides of neck, axillæ and groin.

*Thorax.*—Pericardium, small amount of clear fluid. *Heart* not enlarged, flabby, valves comp. aorta normal. *Lungs* quite normal. No pleural effusion.

*Abdomen.*—Relations of organs natural. No peritoneal effusion. Liver slightly enlarged, early nutmeg. Spleen very little enlarged. Slate col. Caps. Sec. dark pigm. Kidneys nil. Pancreas nil. Bladder normal. Intestines, large intestine full of hard fæces. Parasites, many ankylostomes, very few asc. lumbar, no trichocephalus dispar. Mature *perstans* in mesentery. 4 ♀ portion ♂.

*Brain.*—Dura mater not adherent to the calvarium. Vessels very congested. Longitudinal sinus full of coagulated blood. Pia-arachnoid over the convexities thickened and opaque, no flattening of convolutions. Cerebro-spinal fluid slightly turbid and in excess. On section tissue of brain firm ventricles not dilated. Pituit. body normal, cerebellum, pons, medulla nothing macrosc. Cord not examined.

*Histological Examination.—Brain.* Usual mononuclear exudation under pia and round vessels in interior of brain, much slighter, however, than in the other cases. Capillaries congested.

*Spleen.*—Old malarial pigment.

*Liver.*—Old malarial pigment, cloudy swelling of cells.

*Kidneys.*—Cloudy swelling of epithelial cells.

*Mesenteric Gland.*—Some thickening of capsule, no congestion or dilation of vessels. Lymph. tissue apparently normal.

CASE 4.—Tonnionza. F., 26. Ad. July 18, 1902. Dis. September 30, 1902. Died. *P.M.* Native of Busi, Uganda :—

*Family History.*—Ch. 2. 1 died of S. S. in hospital No. 5, others said to be healthy. B. & S. O. Half-brother l. h.

*Pers. History.*—Brought in from Busi. Nothing else obtainable.

*State on Adm.*—Apparently ill. Tremor of tongue very marked, also in muscles of neck. Arms, fine tremor, legs also. Covered with scabies from head to foot. Lymph. glands in sub-mental region and under jaws. Also in groin, those on rg. side tender. T. 100. P. 86. Glands not very large. No cough or evidence of other disease.

*Circ. System.*—P. 86. Reg. equal. Vol. small, tens. low. V. W. not thickened. A. B., 5th space, 3½ inches from m. l. u. b. 3rd rib, r. b. 1¼ inches rg. of m. l., l. b. 4 inches from m. l. Ausc. M. A. First sound nil. Syst. murmurs at A. A. and P. A., second nil.

*Resp. System.*—18. Reg. equal, shallow. Thorac. abdom. Note res. all over. Br. feeble and faint. Vesic. insp. not prolonged.

*Aliment. System.*—Inspec. nil. Spleen palpable. *Liver* R. D. 5th rib, A. D. 6th, L. B. ¼ inch below C. M. *Spleen* U. B. 7th, L. B. 2 inches below C. M. Ant. to near mid-line.

*Urinary System.*—July 24. Menstruating exam. postponed. August 5. Quant.? Dep. Muc., sp. gr. 1025. No alb., blood, or sugar.

*Lymphatic System.*—Enlarged glands post triangles, nil sub-mental, nil axillæ. Shotty in both groins, more so on right than left.

*Nervous System.*—Very stupid and unintelligent. Heavy silly expression. No marked sleep. No delirium or coma. Will not answer questions well; speech slow and mumbling.

*Cranial Nerves.*—Smell and sight impossible to determine. No paralysis eye muscles; no nystagmus. Pupils equal, mod. dil. React to light and accom. No facial paralysis or of other cranial nerves.

*Motor Functions.*—Motor poor, diminished. Co-ordination impaired. Muscles somewhat flabby. Tremor fine marked in tongue and arms and legs. Weakness of legs, no def. paralysis.

*Sensory Functions.*—Touch unimpaired. Hyperæsthesia all over. Temperature sens. about normal. Resists being handled greatly, and gives short cries of pain. Hyperæsthesia of 5th nerve where branches come out.

*Reflex Functions.*—Plantar, present good, also other superficials. Knee exaggerated both sides, rg. more than left. *Organic.* Passes water in bed, also motions, this however probably due to inability to get out of bed. No ankle clonus. *Gait.* Peculiar, slow, and shuffling. *Throat, nose*, etc., nil.

*Further Notes.*—August 1. A little worse; very dull and stupid. Resists being touched very much. Evident pain menstruating, and passing water in bed. Drowsiness marked.

August 5. Slightly better, very dull and stupid. Eruptions diminishing greatly under sulphur and cleanliness. Knee reflexes still very exaggerated both sides. Spleen punct. 5 P.M. Yesterday and to-day has ceased passing motions in bed, so probably no paralysis of sphincters, only weakness and no proper nursing facilities.

August 13. Q. grs. 5. Mag. sulph. ℥j, twice daily; eruptions much better. Much better again, gets up daily and eats well.

August 18. No change.

August 25. Much worse. Dull apathetic expression. Patient does not get up. Tremor much increased. Knee reflexes exag., especially right side.

August 26. No change.

August 28. A little better.

August 30. No change.

September 2. Better. She is able to get up. Tremor of tongue and hands well marked. Knee reflexes still exag., espec. right side.

September 8. About the same. Med. stopped.

September 11. Tremor in tongue very marked. Also in arms, emaciation becoming marked. Goes about now outside all day.

September 18. Worse again. Very drowsy. Taken to bed. Passing motions in bed. No eruptions now. Emaciation extreme.

September 23. Looks a little better, brighter, does not leave bed now, however. Tremors very marked. Emaciation extreme. Temp. subnormal. Reflexes still sl. exag. about the same.

September 24. Very apathetic. Tremor in tongue and hands very marked. Looks bad.

September 25. Much the same. Lying in bed. Extremities very cold.

September 27. Semi-conscious. Not sleeping, can be roused by stimulation.

September 28. Semi-conscious. Mucus welling from mouth and nose. Very bad. Reflexes still present, but now greatly diminished. Pulse imperceptible at wrist. Very cold, especially extremities. Resists being touched, which apparently causes great pain. Cries out.

September 29. In a state of coma, almost complete; can be roused only with great difficulty. Temp. very low. Motions passed spontaneously. Emaciation marked.

September 30. 8 A.M., complete coma; respirations deep, laboured. Eyes wide open, conj. glazed. Reflexes there still present. Cadaveric smell. 4 P.M., still living. 6 P.M., died.

## Table of Blood Counts.

| 1902. | |
|---|---|
| August 15. | R., 2,700,000; W., 4300; Hb., 62 per cent. |
| August 19. | R., 2,160,000; W., 3750; Hb., 60 per cent.; P. M. N., 52 per cent.; L. M., 30 per cent.; l., 14 per cent.; E., 4 per cent.; T., 0 per cent. |
| September 18. | R., 3,000,000; W., 4300; Hb., 65 per cent. |
| September 23. | R., 3,160,000; W., 4370; Hb., 65 per cent. |
| September 24. | R., 3,000,000; W., 4300; Hb., 64 per cent. |
| September 25. | R., 2,800,0 0; W., 3100; Hb., 64 per cent. |
| September 26. | R., 2,400,000; W., 3120; Hb., 60 per cent. |
| September 27. | R., 2,600,000; W., 3100; Hb., 60 per cent. |
| September 28. | R., 2,720,000; W., 12,500; Hb., 60 per cent. |
| September 29. | R., 3,100,000; W., 26,800; Hb., 64 per cent.; P. M. N., 72 per cent.; L. M., 15 per cent.; l., 13 per cent.; E., 0 per cent.; T., 0 per cent. |
| September 30. | 8 A.M., R., 4,280,000; W., 25,600; Hb., 75 per cent.; 4 P.M., R., 4,680,000; W., 15,600; Hb., 80 per cent. |

*Post mortem*, October 1, 8 A.M.

*External Appearances.*—Extreme emaciation. Many pigmented spots, about the size of a florin, all over body. No signs of the old scabies. No bed sores or eruptions.

*Thorax.*—No pericardial fluid. Heart small. Muscular substance somewhat flabby. Rg. auricle, a lot of p. m. clot, valves healthy; no endocarditis. Aorta healthy.

*Lungs.*—No pleural fluid. R. L., healthy. No congestion; some œdema of base of lower lobe; no bronchitis or pneumonia. L. L., a little œdema and congestion of lower lobe.

*Abdomen.*—No peritoneal fluid. Exam. of fæces just after death showed ova of ascaris, ankylostoma, oxyuris vermicularis, and Bilharzia.

*Liver.*—A few adhesions to diaphragm. Old malarial pigmentation.

*Spleen.*—Enlarged markedly. Capsule slate-coloured. Section pigmented, chronic malarial.

*Kidneys.*—R. Capsule strips easily; substance normal. L. ditto.

*Stomach.*—Nil. Duodenum a few punctiform points; no ankylostomes there, but many in upper part of jejunum. Intestines nil. Rectum, four small ulcerated hæmorrhagic spots small in size, scraping many Bilharzia ova. Embryo adults not found.

*Parasites.*—Ascaris lumbricoides; many ankylostomes; many trichocephalus dispar. Many bilharzia. Ova numerous. *Bladder* healthy. *Uterus* healthy. Vagina, some old inflam. and ulcerative change near vulva.

*Neck.*—Nil.

*Lymph. System.*—Enlarged in all areas, pea to brazil nut, four posterior abdom. ones as large as walnuts, very hard.

*Head.*—Calvarium not thickened. *Dura.* Some congested vessels on upper aspect. No excess sub-dural fluid and no adhesions. Pia arachnoid clear; no excess of fluid. Veins over vertex congested. Very little macroscopic change; other parts of brain no change noticeable; cord not taken out. Lat. ventricles not dilated, contained a little fluid.

*Histological Examination.*—Spleen and liver much malarial pigment. Brain presented same appearance as former cases. Meningeal exudation, and also perivascular infiltration in substance, latter not very marked.

Case 5.—Yaro. M., 35. Ad. July 18, 1902. Dis. September 4, 1902. Died. *P.M.* Native of Buse, Uganda:—

*Fam. Hist.*—F., d.? M., d.? B., l., 40. S. 1, l. h., ch. 0.

*Pers. Hist.*—A labourer, brought in, said to be suffering from Sleeping Sickness.

*State on Admiss.*—Heavy, stupid, unintelligent man, speech slow and thick. Some emaciation, skin dry and harsh, covered with old scabs and sores, pruritus; scratching himself. Enlarged glands, triangles of neck, sub. max., sub-mental, also groins and axillæ. Tremor fine in tongue, also slight in hands, not in legs. T. 99, P. 88, R. 16. Walk typical slow, and pushes feet along ground rather than raising them.

*Circ. Syst.*—P. 100, reg. eq., vol. very small, tens. low, V. W. unthickened. *H.*, u. b., 3rd r., r. b., 1 inch to rg., m. l., l. b. 4 inches to lf., m. l., a. b., 5th, s. p. 3½ inches lf. of m. l. *Aus.* Sounds faint, espec. at base, no murmurs.

*Resp. Syst.*—R. 16, reg. eq., thor. abdom. Insp. nil. Palp. reson. Note all over. Ausc. Vesicular ordinary.

*Aliment. Syst.*—Tongue small, pointed, lips not thick, fur on dorsum. Appetite good. Bowels constip. Abd. nil on insp. *Liver*, u. b., r. d., 6th r., a. d., 7th r., l. b. c. m. *Spleen*, u. b., 8 r., l. b., 12th r. Ant. mam. line, enlarged.

*Urinary Syst.*—Sp. gr. 1018, no alb., blood, or sugar. Straw-coloured. Slight deposit of mucus.

*Skin.*—Rough, dry, harsh, old scratch marks. Covered with old dried-up pustular scabs all over body. Always scratching himself.

*Lymphatic Syst.*—Glands enlarged generally.

*Nerv. System.*—Heavy, dull, stupid, slow speech, imperfect articulations. No delirium or marked tendency to sleep, but drowsy and stupid. *Cranial* nerves, no paralysis evident.

*Motor Functions.*—Muscular nutrition poor, though no marked emaciation. Grip feeble for a man of his age and development. No paralysis. Incoordination impaired distinctly. Romberg's sign present. Other coordinated movements impaired also. Fine tremor tongue and arms, none in legs. No rigidity or contraction.

*Reflex Functions.*—Knee reflexes present. More marked left than right. No ankle clonus. Superficial present, slight.

*Sensory Functions.*—Pain, touch, and temp. present; no hyperæsthesia.

*Special Senses.*—Eyes, pupils equal, mod. dil., react. to light and accom. No nystagmus. No ocular paralysis.

*Further Notes.*—August 14, 1902. About the same, pulse increased in frequency, 120, very small vol. and low tension. Sounds very faint, tremors about the same.

August 25. In the last few days patient has got much worse, much wasted, extreme weakness, bedridden.

August 29. Sub-normal temp., thready pulse, drowsiness very marked; he sleeps also much more than he used, no contractures, no convulsive fits.

August 31. No change.

September 3. Very ill, about dying. Cold, sub-normal temp. comatose. Eyes open, conj. reflex still present, slight. Saliva dribbling from mouth. Unconscious. Emaciation becoming very marked. No fits. Pulse thready, almost imperceptible. Motions passing spontaneously. Jaundice of conj. appeared yesterday.

## Table of Blood Counts.

| 1902. | | | |
|---|---|---|---|
| September 11. | R., 3,875,000; | W., 8,700; | Hb., 79 per cent. |
| September 18. | R., 4,360,000; | W., 10,000; | Hb., 85 per cent. |
| October 9. | R., 4,200,000; | W., 8,000; | Hb., 84 per cent. |

September 4. *Died about* 6 A.M. *Post-mortem examination at* 10 A.M.

*Ext. Appearances.*—Body considerably emaciated. Skin dry and harsh. Pig. scars all over body, especially legs; no bed sores. Jaundice in conjunction.

*Thorax.*—No pleural effusion. Old adhesions upper part of left lung. *Left* lung healthy. *Right* lung rather deeply congested. *Pericardium*, no fluid. *Heart* substance, degenerated, flabby. Valves competent, aorta healthy.

*Abdomen.*—Fat on section, very yellow icteric staining.

*Spleen.*—Enlarged, perisplenitis, old. Capsule slate-coloured. Section, congestion and pigmentation.

*Liver.*—Enlarged, adherent to diaphragm; old perihepatitis. Section, congestion, and fatty infiltration.

*Gall Bladder.*—Much distended. Duct blocked by a soft dark coloured gall stone, also several smaller ones. Contents of gall bladder black, grumous liquid. Opening into duodenum patent.

*Stomach.*—Hour-glass constriction, enlarged. Inner surface no old ulceration. Part opposite constriction thicker than rest. Duodenum and intestines contain much slimy mucus, otherwise normal. Cæcum loaded with hard fæces. *Parasites.* Ankylostomes plenty, also Trichocephalus dispar; no Lumbricoides.

*Bladder.*—Nothing to note.

*Kidneys.*—R. capsule strips easily. No inflam. L. ditto.

*Pancreas.*—No congestion, normal in consistence.

*Mesentery.*—Numerous enlarged glands, from pea to hazel nut.

*Neck.*—Tonsils slightly enlarged. Larynx healthy. Glands of a general small size. Sub-mastoid. Sub-maxillary. Sub-mental, and in the triangles. No suppuration or breaking down. Thyroid, nothing to note.

*Head.—Scalp* fat, markedly yellow, bile stained. *Calvarium* normal consistence. *Long Sinus* contains some fluid blood, and also a dark-red clot. *Dura Mater.* Slightly adherent in places in middle line, otherwise not. Slight excess of fluid. Pia arachnoid can be stripped easily without erosion. Vessels on vertex much congested. Sub-pial fluid in excess, not turbid, markedly bile stained. At places recent lymph. No pus or suppuration. Convolutions no flattening and no adhesion of Pia in sulci. Appearance of Pia arachnoid membrane somewhat opaque. A considerable amount of fluid in ventricles. Pons, Pituitary body, Medulla and Cerebellum, nothing of note macroscopically. *Cord.* Dura not adherent. No congestion of vessels. Pia strips readily. Substance of cord apparently healthy macroscopically.

*Histological Exam. Brain.*—Usual exudation on meningeal surface, slight round vessels in substance.

*Pons.*—Slight exudation round vessels.

*Medulla.*—Similar exudation, and about same in amount.

*Spinal Cord.*—Cervical region. Meningeal exudation, also in some vessels in grey matter, not in white. Other regions similar.

*Sciatic Nerve.*—Degeneration ?

*Cerebellum.*—Usual exudation in sulci and on outer surface.

*Stomach.*—Catarrh of mucous membrane. Wall hypertrophid, especially sub-mucous layer.

CASE 6.—Kasokabilia. M. 32. Ad. July 18, 1902. Dis. August 31, 1902. Died. *P.M.* Native of Buse, Uganda :—

*Fam. Hist.*—F. l. h. M. d. B. 2. l. h. S. O. Ch. 2. d. ?

*Pers. Hist.*—Labourer. Prev. ill, nil. ex. s. pox.

*State on Ad.*—Big burly negro, pitted with old small-pox marks. Heavy expression, not drowsy. Very marked tremor in tonguo and arms, legs fine in character. Walk impeded, puts feet down all right, rather shuffling. Old scars and dried spots, one large scar on right buttock, one on left knee, many on legs. Old spots limbs and body. Glands small in both triangles, also slight under jaw. Shotty and hard enlarged glands both groins. Skin rather dry and harsh, lustreless. Eats well. T. 102° F. P. 100. R. 20.

*Circ. System.*—P. 100, Rg. equal, small vol., low tens., V. W. unthickened. a. b. 5th space, 3 inches from m. l. u. b. 3rd rib, r. b. 1 inch rg. m. l. l. b. 3½ inches from m. l. at 4th rib. Sounds faint, no murmurs, better heard base than apex.

*Respir. Syst.*—R. 20 equal reg., thoracic abdominal. V. F. normal. Note resonant all over breathing, ordinary vesicular, no prolongation expiration.

*Aliment. System.*—Tongue flabby, dirty fur. Bowels constipated. Abd. nothing on inspection. *Liver*, u. b., r. d., 5th, a. d. 6th l. b., c. m. *Spleen*, u.b. 7th, l. b. 12th ant c. m. in mam. line.

*Urinary System.*—Sp. gr. 1012, no alb., blood or sugar, deposit nil. No subjective symptoms.

*Skin.*—Old scars buttocks, limbs, knee, and lower leg. Skin harsh and dry, no pruritus.

*Lymph. System.*—Small enlarged glands in triangles of neck and sub-max. hard in consistence ; also in both groins and axillæ.

*Nervous System.*—Not specially dully or drowsy. Answers questions intelligently. *Cranial Nerves.* No paralysis in evidence.

*Motor Functions.*—Legs, muscles flabby. No marked emaciation of any part of body. No incoordination. No Romberg's symptom. Tremor very fine marked in tongue, hands, arms, and legs. Very noticeable feature of case on inspection.

*Sens. Functions.*—No impairment, touch, temp. or pain sense. Pain sense increased, some hyperæsthesia in limbs.

*Reflexes.*—Superficial present. Knee equal. Slightly diminished. No ankle clonus.

*Special Senses.*—Eye, no ocular paralysis. Pupils equal, mod. dilated, react to light and accommodation. Smell unimpaired, hearing also.

*Further Notes.* July 25. Tremors very marked, increased, eats well and gets up daily and goes outside.

August 1. Much the same, tremors still very marked, tongue, arms, body and legs.

August 7. Worse. Taken to bed to-day and has not been up. Still eats well, tremors as before.

August 12. Never out of bed since 7th, passed water in bed to-day for first time, due to weakness and inability, probably did not ask for urine dish. Very weak and getting rapidly worse.

August 13. Passed water and fæces in bed to-day. *Circ. Syst.* A. b. not palp. borders as before. Sounds almost indistinguishable. P. 130, pract. no vol. or tension, almost impossible to feel. *Resp. System.* Nothing in chest, reson. note at bases and no prolongation in expiration or auscultation.

*Alim. Syst.*—As before.

*Nerv. Syst.*—For last six days tremors have increased and are now so marked as to shake bed clothes. Not coarse, still fine in nature. Some rigidity of neck, and painful when touched. Contracture of legs, flexion of thighs on abd. and legs on thighs appeared six days ago and is now marked, cannot straighten them, and cries out with pain when one tries to do so. Reflexes difficult to obtain owing to contracture, but still present.

*Sens. Functions.*—Some hyperæsthesia about neck and marked pain in legs. Temp. sense unimpaired.

*Spec. Senses.*—Eyes wide open. Pupils equal, mod. dil., react to light and accom. Patient very weak and apparently near end, still eats of own accord.

August 14. Still living. Sordes on gums and teeth, foul smell, very weak. Urine drawn off by catheter, a little blood from urethra, which accounts for resulting slight trace of albumin. No sugar.

August 15. Slightly better.

August 16. About same, extremities cold, pulse very feeble, passing motions in bed.

August 18. Still living. Tremors excessive. Eyes wide open, no paralysis definite. Passing motions in bed.

August 19. Pulse imperceptible. Extremities cold. Much purulent discharge welling out from nose, passing motions in bed. Marked leucocytosis suddenly appeared to-day.

August 23. Great fall in temp., extreme weakness. A skin eruption with large blebs on the left forearm, liquid of blebs clear, containing very few leucocytes.

August 24. No change.

August 25. The contents of the blebs has become purulent, dressing with sublimat.

August 27. General conditions much worse. Pulse almost imperceptible, respiration frequent and shallow, less tremor.

August 28. No change.

August 29. No change.

August 30. Pulse imperceptible, respiration shallow and very frequent (44), extremities cold.

August 31. The patient died suddenly about 5 A.M.

## Table of Blood Counts.

| | |
|---|---|
| 1902. | |
| August 14. | R., 3,812,000; W., 5400; Hb., 75 per cent. |
| August 15. | R., 4,000,000; W., 6870; Hb., 78 per cent.; P. M. N., 56 per cent.; L. M., 28 per cent.; l., 12 per cent.; E., 1 per cent.; T., 3 per cent. |
| August 19. | 4 P.M. R., 5,200,000; W., 33,125; Hb., 90 per cent.; P. M. N., 70 per cent.; L. M., 20 per cent.; l., 8 per cent.; E., 0 per cent.; T., 2 per cent. |
| August 20. | 4 P.M. R., 6,200,000; W., 22,000; Hb., 100 per cent.; P. M. N, 70 per cent.; L. M., 17 per cent.; l., 13 per cent.; E., 0, per cent.; T., 0 per cent. |

*P.M.* 10 A.M., 5 *hours after death.*—Body very emaciated. Bed-sores on trochanters. Glands palpable, neck, axilla, groin.

*Thorax.*—Relations of organs natural. Pericardium appears normal, a very small amount of clear fluid. *Heart* not enlarged. Flabby, much fat. Valves competent. Nothing to be noted in the aorta. *Lungs* normal, no pleural effusion.

*Abdomen.*—Relation of organs natural. No peritoneal effusion, mesenteric glands enlarged. *Liver*, not enlarged, normal. *Spleen*, slightly enlarged, capsule slate-coloured on section, very dark pigment. *Kidneys*, nil. *Pancreas*, nil. *Bladder*, nil. *Intestines*, large intestine full of hard fæces. *Parasites*, many ankyl., few asc. lumbr., no tric. dispar.

*Brain.*—Calvarium dense and hard, symmetrical. Dura mater not adherent, vessels very congested. Longitudinal sinus full of coagula. Pia arachnoid over the convexities opaque, can be stripped without erosions, no flattening or wasting of convolutions, cerebro-spinal liquid a little turbid and in excess. On section tissue of brain from ventricles not dilated. Pit. body normal. Cerebellum, pons, medulla appear normal. Cord looks healthy macroscopically.

*Histological Examination.*—Brain. Picture of usual Sleeping Sickness, meningo-encephalitis. Infiltration of small cells under pia, mononuclear lymphocytes spreading down the sulci. Vessels in substance of brain packed round with a layer of mononuclear leucocytes. No other changes visible. Similar changes in pons, medulla, and cord.

Case 7.—Msavika (No. 1). F. 12. Ad. July 31, 1902. Dis. September 16, 1902. Died. *P.M.* Native of Buse, Uganda :—

*Faml. Hist.*—Unobtainable.

*State on Adm.*—Peculiar-looking child. Standing, fine tremor seen. Left forearm flexed at elbow and held with extensor surf. uppermost, drop wrist. Other arm normal as to position, fine tremor in both. Walks peculiarly, dragging limbs, also walks with toes of left foot in air. This foot badly infected with jiggers, probably explains position. Tongue, fine tremor. History of headache, but none now. Enlarged glands in post. triangle of neck and under jaws, also inguinal glands shotty in consistence. Skin smooth, no pruritus or eruption of any kind. Passes motions normally. Some rigidity of neck, especially on left side.

*Circ. System.*—P. 98. Reg. equal. Vol. small, tens. low, V. W. nil. A. B. 6th sp. 3½ inches from m. l., u. b. 3rd rib, l. b. 4 inches from m. l., r. b. 1 inch to rg. m. l. Ausc. Sounds distinct, no murmurs.

*Resp. System.*—R. 16. Reg. equal. *Aus.* Vesicular, note reson. all over.

*Alim. System.*—Insp. nil. *Liver* u. b., r. d. 5th, a. d. 6th, l. b., c. m. *Spleen* u. b. 7th, l. b. 12th r. *Bowels* constip., motions passed normally.

*Urinary Syst.*—Quant. ? Dep. muc., sp. gr. 1024. No alb., blood, or sugar.

*Nervous Syst.*—Rather frightened-looking child. No drowsiness or marked sleep tendency. No coma or delirium. Speech unaffected. *Cranial nerves*, no paralysis evident. *Motor Functions*, marked wasting and feebleness of leg muscles and arm. Grip feeble. Fine tremor tongue, arms and legs. Left arm held in peculiar position, flexed at elbow and wrist hangs. Can be straightened, but some difficulty (contracture). No incoordination.

*Sensory Funct.*—Unimpaired generally. Some hyperæsthesia locally in legs and arms. No pains in head. Pain complained of in rg. side over kidney.

*Reflexes.*—Superficial slightly diminished, knee present ordinary, rg. increased left. No ankle clonus. Organic nil.

*Special Senses.*—Eye, no ocular paralysis. Pupils equal contract to light and accommodation. No nystagmus.

*Skin.*—No eruptions or pruritus, skin smooth and glossy.

*Lymphatic Syst.*—Enlarged glands, neck shotty in ant. and post. triangles. Submental, groins also.

*Further Notes.*—August 2. No change.

August 5. Pain over rg. side gone. No headache. Tremors dist. less. Left knee reflex distinctly exagg., rg. very slightly increased. No ankle clonus.

August 13. Quin. 5 grs. Mag. sulph. ʒ j. twice daily.

August 14. Not so well, tremors increased again. Left arm more contracture. Tendency to contracture in limbs.

August 25. Much worse. Bedridden. No drowsiness.

August 30. Patient appears in great pain, crying out frequently, half unconscious. Does not answer to questions.

August 31. A little better, does not cry any more.

September 2. Better.

September 3. No change.

September 5. Much worse. Every now and again gives sharp cries. Tremors much more. Does not leave bed. Feeds with difficulty. Passing motions in bed.

September 8. Worse. Lies in bed. Groaning and crying out. Tremors very marked. Emaciating. Eyes wide open. Passing motions spontaneously.

September 11. Very bad. Lying in a semi-comatose condition, with eyes wide open. Contracture of legs excessive, calf on thighs and latter on abdomen. Contracture in left arm more marked than when came in. Emaciating rapidly. Large deep bed sore appeared on left trochanter with slough in centre. Does not answer questions or appear to hear when spoken to. Has stopped crying out. Motions passed in bed. Nothing cardiac or respiratory.

September 15. Very ill. Mucus welling from mouth. Semi-comatose, does not understand. 6 P.M. comatose, cold extremities, sub-normal temp.

September 16. Died 3 P.M. No fits, continued in state of coma.

## Table of Blood Counts, &c.

| 1902. | |
|---|---|
| August 20. | R., 3,080,000 ; W., 4900 ; Hb., 66 per cent. ; P. M. N., 54 per cent. ; L. M., 14 per cent. ; l. 26 per cent. ; E., 3 per cent. ; T., 3 per cent. |
| September 12. | R., 2,330,000 ; W., 5000 ; Hb., 56 per cent. ; P. M. N., 61 per cent. ; L. M., 18 per cent. ; l., 15 per cent. ; E., 4 per cent. ; T., 2 per cent. |
| September 15. | R., 1,630,000 ; W., 13,120 ; Hb., 50 per cent. |

*Post Mortem.*—Died 3 P.M. *P.M.*, 4 P.M., September 16.

*Ext. Appearances.*—Much emaciated. Large bed sore over left trochanter, Another one over upper part left buttock. Several smaller ones over dorsal region of back, left side. Skin dry and scaly, no old scars, no scratch marks, no eruptions.

*Chest.*—Pericardium, no fluid. Valves of heart competent. Musc. substance not degenerated. Pale in colour. *R. Lung* congestion and some consolidation of lower lobe early. Still floats in water. Substance of rest of lung, very little blood, some œdema. *L. L.* no congestion, bloodless, some œdematous fluid. Pleural cavity no fluid, many old adhesions.

*Abdomen.*—Old adhesions round spleen and liver. Omentum adherent to liver. No fluid. *Liver*, ordinary size. Tissue normal in appearance. *Gall Bladder* contained a little bile, green colour. *Spleen* enlarged slightly, pigmented substance and capsule. Pancreas and kidneys normal. *Stomach* normal. Intestines full of hard fæces. *Parasites*, no ankylostomes. Many trichocephalus and ascaris lumbricoides. Uterus and bladder nil. All organs very anæmic.

*Neck.*—Thyroid and larynx nothing, enlarged gls. hard in consistence in all neck areas, groins, femoral and other regions.

*Brain.*—Calvarium rather thin. Dura not adherent, no fluid. Sub-dural vessels congested. Sinus, some dark black blood. Pia-arachnoid not opaque, strips easily, practically no excess of fluid. Substance of brain and other parts show practically nothing macroscopically. Cord not examined. No excess fluid in ventricles.

*Histological Examination.*—Spleen, old malarial pigment. Pig. leucocytes and a few malig. rings. *Lung*, pneumonia rg. lower lobe. *Brain*, lymphocytic exudation very marked round vessels of brain, motor area. Medulla, very great infiltration round vessels, also exudation on meningeal surface. *Pons.* similar. Case presents features of an excessive infiltration round vessels of central nervous system.

CASE 8.—Msavika. F. 35. Ad. July 31, 1902. Dis. September 24, 1902. Died. *P.M.* Native of Busi, Uganda :—

*Family History.*—F. ? S. 1. L. Ch. 0.

*State on Ad.*—Stupid expression, dull and apathetic, fine tremor in tongue, none in arms. Walk slow and unsteady, tendency to cross feet. Glands enlarged post triangles neck, also both groins, size of beans, none sub-mental. Harshness of skin on arms, on body soft. No pruritus or eruptions.

*Circ. System.*—P. 90. Reg. equal, vol. small, tens. low, V.W. unthickened. a.b. not pal., u.b. 2nd rib lower bord. R.B. 1 inch to rg. m.l. l.b. 5 inches to l. of m.l. Sounds at apex very faint, no murmur. More audible, but still feeble at base, no murmurs.

*Resp. Syst.*—R. 18. Reg. eq. Thor. abdom. No cough. Note reson. Ausc. nil.

*Alim. System.* Liver u.b. R.d 5th, a.d., 6th l.b., e.m. *Spleen*, palp. u.b. 7th, l.b. just below e.m. *Bowels* constip.

*Nerv. System.*—Excessively stupid. Lethargic and apathetic, no marked tendency to sleep.

*Cranial Nerves.*—No paralysis.

*Motor Funcs.*—Slight wasting legs, consistence flabby. Fine tremor, tongue, hands, arms and legs. No incoordination. No paralysis, walk heavy, but does not stagger.

*Sens. Functs.*—Unimpaired, touch pain and temp. sense not diminished or exaggerated. *Reflexes*, superficial present well marked. Knee reflexes both increased, equal slight. Organic not impaired. Ankle clonus nil.

*Special Senses.*—Eye. Pupils equal, mod. dilated, react to light and accommodation. No nystagmus. No ocular paralysis.

*Skin.*—Harsh and dry, no papular eruptions.

*Lymph. Syst.*—Glands enl. both triangles neck, groins, sub-maxillary, sub-mental, and axillary.

*Urinary Syst.*—Sp. gr. 1015. No alb., bl., or sugar.

*Further Notes.*—August 5. Stupidity increased. Doses a good lot, obeys when told to do anything, very slowly. Appetite very good.

August 13. Q. grs. 5. Mag. sulph, ʒ ij., twice daily.

August 25. Very drowsy appearance. Patient has lost flesh in last days. Knee jerk increased, especially on left side.

September 3. No change.

September 5. Emaciation becoming very marked. Tremors increased. Skin very dry, harsh, pruritus, though no eruptions. Much feebler, walk tottering and difficult.

September 17. Took to bed yesterday, has become bedridden. Very drowsy and stupid, emaciation proceeding rapidly.

September 23. Completely bedridden. Passing motions in bed. Emaciation extreme. Knee reflexes slightly increased, left more than right. L.P. done.

September 24. Very ill. Pulse imperceptible at wrist. R. 36, deep and laboured. Conj. glazed. No pneumonic signs.

10 A.M. Skin extraordinarily rough and thick. Very cold. Temp. sub-normal.

3 P.M. Died suddenly. No fits or convulsions.

## Table of Blood Counts.

September 17. R., 2,620,000; W., 7800; Hb., 62 per cent.
September 23. R., 1,950,000; W., 13,000; Hb., 42 per cent.

*Post-Mortem Examination. Died* 3 P.M. *P. Mortem* 4 P.M.

*Ext. Appearances.*—Body cold; very much emaciated. No old scars or bed sores. Skin very rough and harsh all over body. Some old scratch marks. No eruptions of any sort.

*Thorax.*—*Pericardium,* no fluid. *Heart.* Some p. m. clot in rg. ventricle. Muscular substance pale, firm in consistence, no degeneration. Valves competent. No atheroma or disease of those or aorta. *Lungs.* No pleural fluid, a few adhesions, old, on rg. side. *Lung, R.* Pale and bloodless. No congestion or pneumonia. A few collapsed portions near pleura. *Lung, L.* Ditto to rg.

*Abdomen.*—No peritonitis. *Liver.* Not enlarged, section normal. *Spleen.* Slightly enlarged, section dark red, capsule not markedly slate-coloured.

*Stomach.*—Some thickening of middle layer at pyloric orifice. Mucous membrane healthy.

*Pancreas.*—Nil.

*Kidney, R.*—Very small. Capsule strips with some difficulty. Substance pale, no inflam.

*Kidney, L.*—Ditto to R., and of same size.

*Uterus and Ovaries.*—Nil.

*Bladder.*—Distended, mucous memb. ordinary.

*Intestines.*—Contained somewhat liquid dark coloured fæces. Appendix normal-Parasites, plenty Ankylostomes, no trichocephalus or ascaris lumbricoides. Mesenteric glands, small enlarged. No mature filariæ found in mesent.

*Neck.*—Slightly enlarged left tonsil. Tongue, larynx and pharynx all healthy.

*Lymphatics.*—General enlargement. Glands discrete. Small in size, average a small bean, hard in consistence, no suppuration anywhere.

*Head.*—Calvarium unthickened. Sub-dural fluid much in excess. L.S. some dark blood clot. Veins over vertex full of dark blood, congested. Some capillary congestion also. Pia arachnoid not opaque, clear, but markedly adherent, especially in sulci. Some excess of its fluid at places. Causes a little erosion at places when stripped. Excess of fluid in ventricles, and both lateral ones slightly dilated. Pituitary body, pons, medulla and cerebellum nil macroscopically. Spinal fluid drawn off by lumbar puncture in excess. Cord not removed.

*Histological Examination. Spleen.*—Very old malarial pigment, no parasites.

*Brain.*—Presents ordinary changes, not excessive Mononuclear leucocyte exudation round vessels and on meningeal surface of brain. Other organs, some leucocytic infiltration in substance of heart. Liver cloudy, swelling.

CASE 9.—Kagawa. F., 18. Ad. August 11, 1902. Native of Busoga:—

*Fam. Hist.*—F. d. k., M. d. fever, B. m., l. d., sl. sickness. Ch. 0.

*Pers. Hist.*—Six months ill. Began then to be drowsy, had to be awakened in mornings. No illness before.

*State on Adm.*—Good-looking woman, fairly intelligent, slight tremor of tongue, none in arms. Not drowsy. T. 103, P. 130, R. 28. No emaciation. Glands small in neck, all areas, specially on left, also sub-maxillary region. Groins, big glands both sides, some as large as small walnut. Slight drowsy and heavy expression. No rash or pruritus.

*Circ. Syst.*—P. 130, reg. equal, vol. small, tension low, V. W., unthickened; a. b. palpable, 5th sp., 3½ inches from m. l., u. b. 3rd rib, l. b. 4 inches from m. l., r. b. 1 inch rg. m. l. Ausc. Sounds distinct, no murmurs.

*Respir. System.*—28, reg. equal, sl. hoarseness. V. F. not increased. Note resonant. Sounds normal.

*Alim. System.*—Tongue dirty, heavy white fur. Appetite good. Bowels constipated. Nothing on inspec. Stomach not enlarged. *Liver* u. b., r. d. 6th, a. d. 7th, l. b. c. m. *Spleen* u. b., 9th r., l. b. 11th.

*Urinary System.*—No subjective symptoms. 1018. No alb., blood, or sugar. Deposit of phosphates.

*Skin.*—No pruritus or eruptions. Skin soft in consistence.

*Lymphatic Syst.*—Large groin glands, hard small glands in all triangles of neck, also sub-max. and sub-mental.

*Nervous Syst.*—Slight drowsiness. Cranial nerves. No paralysis. Trigeminal points not painful.

*Motor Functions.*—No wasting of muscles. Leg muscles flabby. Fine tremor only in tongue. No paralysis. Incoordination nil. Sometimes fine tremor in hands. Walk, no marked peculiarities.

*Sens. Functions.*—Pain sense unimpaired, touch and temp. sense not abnormal.

*Reflexes.*—Superficial present, ordinary. Knee reflexes absent. Ankle clonus nil.

*Special Senses.*—Eye, no paralysis, pupils equal, mod. dil., react. to light and accom.

*Further Notes.*—August 12, 1902. Night temp. 104. Q. 5 grs. Mg. Sulph. ʒ ij. twice daily.

August 13. Night temp. 103·8.

August 14. Temp. keeping up, complaining of splenic pain, and also pain in stomach. Mixture stopped to night, *e.g.*, only one dose to-day. Sp. not palp., U. B. 8th r., not tender; l. b. 12th rib, *e.g.*, some slight increase in size since 11th.

August 19. Complaining of pain in chest and over spleen. Bl. exam., no parasites (malarial).

August 25. Temperature very high, intermittent. Patient complaining of muscular pains in the legs. Heart, base diam. enlarged. Loud, rather harsh systolic murmur, with point of maximum intensity at the apex. Sounds accentuated in the pulmonary area.

August 28. No change in general conditions. The murmur is less harsh.

September 2. T. decreased, much better.

September 8. Distinctly changing for the worse. Tremor now in hands. Losing flesh.

September 23. Much better again. Tremor in tongue very slight. None in hands.

September 29. Much the same. Goes about by day.

October 5. A little worse. Some increase of wasting, not markedly stupid. Goes about by day. Appetite good.

October 10. About the same.

October 28. No change. No emaciation. Looks well. Appears to have gained weight. Still fine tremor in tongue, however; none in hands. Intelligent.

## Table of Blood Examinations and Counts.

| | |
|---|---|
| 1902. | |
| August 12. | No malarial parasites. |
| August 19. | R., 4,320,000 ; W., 8400; Hb., 60 per cent.; P. M. N., 62 per cent. ; L. M., 14 per cent. ; l., 16 per cent. ; E., 8 per cent.; T., 0 per cent. No malarial parasites or pigmented leucocytes. |
| September 18. | R., 4,480,000; W., 7000; Hb., 70 per cent. |

CASE 10.—Basibi. M., 35. Ad. August 13, 1902. Left hospital August 25, 1902. Native of Laji. Sesse group :—

*Fam. Hist.*—F. d. stomach. M. l. h. B. 2 l. h. 1 died sl. sickness. S. l., swollen arms and legs. Ch. O.

*Pers. Hist.*—Labourer. No prev. illness.

*State on Ad.*—Brought in by chief. 20 days ago was noticed to walk peculiarly and would not work as usual, reputed to be drowsy. Heavy, dull, apathetic looking man. Drowsy appearance. Large heavy flabby underlip, tongue very marked tremor. Fine tremor in hands. T., 101. P., 95. R., 20. No emaciation; strong built man ; protuberant abdomen ; glands nil in triangles, slight under left jaw. Marked in groins, also in femoral group, on left side one soft and rather boggy. Skin harsh and dry. No pruritus or eruptions. Soft sores on foreskin of penis.

*Circ. System.*—P., 95. Reg. equal. Vol. mod. tens., sl. increase. V. W. thickened.

*Heart.*—Epigast. pulse. A. b. not palp., u. b. 3rd r. R. B. $1\frac{1}{4}$ inches r. m. l., l. b. at 4th rib, 4 inches to l. of m. l. Aus. First redup. 2nd ordinary. Slightly faint at base.

*Resp. System.*—R. 20, equal, reg. V. F. not impaired. Note resonant all over. Sounds. Vesicular all over. No prolongation of expiration.

*Aliment. Syst.*—Tongue large, soft, and flabby; dirty white fur; lower lip hanging and flabby. Abd. protuberant. Bowels constip. *Liver* u. b. 6th and 7th. l. b. $\frac{1}{4}$ inch below c. m. *Spleen,* u. b. 7th, l. b. 11th ant. not enlarged.

*Urinary System.*—Sp. gr. 1026 ; no alb., blood, or sugar. No deposit.

*Skin.*—Harsh and dry ; a few old spots on back.

*Lymphatic Syst.*—As in state on admission.

*Nervous System.*—Distinctly drowsy, stupid heavy appearance. No cranial nerve paralysis.

*Motor Functions.*—Leg muscles somewhat flabby, not wasted. Incoordination nil. Fine tremor in hands ; slight tremor in tongue.

*Sens. Functions.*—Pain, tem. sense and sensation ordinary.

*Reflexes.*—Superficial present diminished ; knee reflexes absent; no ankle clonus. Walk slow, but not typical.

*Special Senses.*—Eye, no ocular paralysis. No nystagmus. Pupils equal, somewhat contracted, react slight to accommodation, not to light, very sluggish. Smell unimpaired.

*Further Notes.*—August 14. Soft sores on penis, no bubo.

August 20. No change.

August 23. Patient has lost flesh; not much drowsiness. Tremor of tongue increased.

August 25. Patient left hospital without permission, and disappeared.

The case is interesting on account of the eye symptoms.

CASE 11.—Busoki. M. 40. Ad. August 1, 1902. Dis. September 5, 1902. Died. *P.M.* Native of Buwaya, Uganda:—

*Fam. Hist.*—F. d. fever, M. d. stomach, B. l. l. h., s. l. l. h. Ch. O.

*Pers. Hist.*—Labourer.

*State on Admission.*—Comparatively intelligent looking, not stupid. Not drowsy, no marked tendency to sleep. Not emaciated. Enlarged glands generally small only in neck. Walk typical, does not raise foot, pushes it, shuffling gait. Speech not slow, answers questions quickly. Fine tremor tongue, none in arms. Skin, soft, smooth, no pruritus or eruptions.

*Circ. Syst.*—P. 90. Reg. eq. Vol. small, tens. slight increase. V.W. slightly thickened.

*Heart.*—A. B. 5th sp., 3½ inches from m. l., u. b. 3rd r., l. b. 4 inches from m. l., r. b. 1 inch to r. m. l., ausc. redup. first sound, second ordin., no murmers.

*Respir. Syst.*—R. 16 equal, reg. cost. abdomen, note resonant all over, breathing ordin., vesic., no prolong. expiration No cough.

*Alim. Syst.*—Tongue, medium size. Some bluish-black pigmented spots on dorsum and frenum. No fur. Abd. nil inspec. *Liver*, u. b. r. d. 5th, a. d. 6th, l. b. c. m. not palp. *Spleen*, u. b. 8th, l. b. 12th, ant. at mam. line, not palpable. Stomach not enlarged. Bowels constipated. Fæces. Many bilharzia ova.

*Urinary System.*—Sp. gr. 1020. Slight deposit of mucus. No alb., bl., or sugar.

*Lymph. Syst.*—Chain of small glands in post. triangles, none in groins or submental, some on sub-max. group.

*Integ. System.*—Some dryness of skin, arms and legs and some pruritus in latter. Skin of trunk, smooth.

*Nerv. System.*—Not stupid, fairly intelligent. No cranial nerve paralysis.

*Motor Functions.*—Consistence of muscles diminished. No paralysis or wasting. No incoordination. No R.'s symptom. Grip and muscular power good. Fine tremor, very slight only in tongue.

*Reflex Functions.*—Superfic., present, slight, knee exagg., rg. ordinary, no clonus, organic normal.

*Sens. Functions.*—Touch, temp. and pain sense unimpaired, no hyperæsthesia or pain over trigem. points.

*Special Senses.*—No ocular paralysis. No nystagmus. Pupils eq., somewhat contracted, react perfectly to light and accom.

*Further Notes.*—August 14. About the same, little change.

August 27. In the last few days an almost sudden change. Extreme weakness, patient is not able to get up from bed, much wasted. Temperature very low, pulse almost imperceptible.

August 29. No change.

September 2. Drowsiness much increased, pulse imperceptible, cold extremities.

September 3. On the point of death, absolutely comatose, only sign of life a slight conj. reflex, temp. sub-normal. Very cold and clammy. No contraction muscles of neck. Saliva dribbling from mouth. Motions passed in bed. No pruritus or eruptions. Skin of trunk still smooth. Reflexes leg gone.

September 4. In a moribund condition.

September 5. Died at 5 A.M. in a state of unconsciousness. No fits, temp. sub-normal.

## Table of Blood Counts.

1902.

August 14. R., 4,400,000; W., 5600; Hb., 85 per cent.
September 4. R., 5,580,000; W., 9500; Hb., 92 per cent.

*P.M. at* 10 A.M.

*Ext. Appearances*—Body not very emaciated. Skin smooth on trunk. Some dryness over arms and legs. No scratch marks or any eruptions or old scars.

*Chest.*—Pericardium, a small amount of clear straw-coloured fluid. Heart rather small, consistence of muscle good. All valves competent. No endocarditis. Aorta healthy.

*Lungs.*—No pleural effusion or old adhesions. Some small glands at root about size of pea. *R. Lung.* Generally œdematous. Upper lobe, œdema, congestion and bronchitis. *Middle*, œdema, some congestion, no pneumonia. *Lower* ditto to middle. *L. Lung.* Upper lobe, œdema, congestion, and bronchitis. *Lower lobe*, œdema and congestion.

*Abdomen.*—*Liver* enlarged, nodular on surface. Some adhesions below it and rg. kidney. *Section* pigmented and full of white fibrous masses, which follow the line of the vessels very markedly. Some of these large areas, some small.

*Gall Bladder.*—Not distended, contained a little greenish bile.

*Spleen.*—Enlarged. Old adhesions. Slate-coloured capsule, section darkly pigmented. Much old malarial pigment, no parasites.

*Kidneys.*—*R.* Capsule strips with difficulty. Cortex not diminished. Substance pale, fatty. *L.* Much larger than right. Capsule strips with difficulty, pale in substance, also fatty.

*Pancreas.*—Very hard in consistence. A fibroid change round vessels similar to that in liver.

*Bladder.*—Normal.

*Stomach.*—Dilated, large, nothing to note in interior.

*Duodenum.*—Mucous membrane congested.

*Jejunum and Ileum.*—Congested.

*Cæcum.*— Congested mucous membrane. Some liquid fæces.

*Parasites.*—A few ankylostomes, no ascaris or trichocephalus. Glands in mesentery enlarged, size of small peas. No mature F. perstans found. 40 adult Bilharzia in mesentery.

*Neck.*—Thyroid and larynx normal. En. glands in all neck areas, small in size, hard. Size of peas.

*Head.*—Calvarium not specially dense. Dura mater, distinctly adherent along middle line. L. sinus, a little coagulum and fluid blood. Pia arachnoid opaque. Sub-pial fluid in excess, clear, pale straw-coloured. No suppuration. Membrane strips without eroding brain surface. Vessels much congested. Section of brain, nothing apparent. Left ventricle much dilated and full of clear straw-coloured fluid. R. lat. ventricle not distended, some fluid. Pituitary body small in size. Nothing macroscopically to be seen in pons medulla and cerebellum. Base of brain presents same picture as vertex. Pia arachnoid opaque with excess of fluid, especially over sulci.

*Cord.*—Excess of fluid. Congestion of vessels. Dura not adherent: Glands: epitrochlear glands both size of small peas. Groin glands also enlarged about same size.

*Histological Exam.*—Old malarial pigment liver and spleen, no parasites.

*Brain.*—Very marked leucocytic exudation mononuclear round vessels in substance, also in the sulci and under pia generally.

*Cord.*—Similar change to brain quite as marked. Infiltration under pia and very marked round the vessels in grey and white matter of the cord. *Nerve root.*—Medullary substance round axis cylinders of nerves in cauda equina degenerated.

*Liver.*—Much malarial pigment. Dense masses of old scar tissue. Old gummata.

*Pancreas.*—Some increase in fibrous tissue at places.

CASE 12.—Zenwala. M., 45. Ad. August 18, 1902. Dis. October 18, 1902. Died. *P.M.* Native of Entebbe, Uganda :—

*Fam. Hist.*—F. d.? M. d.? B. 6, 5 l. h., 1 sus. of early s. s. S. l. l. h. Ch., l. l. h.

*Pers. History.*—Fisherman. Lives a lot on lake in his avocation. Comes from hill No. 2, where a lot of native huts are. Lives on bananas chiefly.

*Pres. Illness.*—Headache of late. People who brought him say he has been ill for some time, two years? this undoubtedly incorrect. Within last month has suffered intermittently from headache, pain in chest, legs, and feet. Has been doing no work lately, but just lying about.

*State on Ad.*—Heavy, stupid looking man, not intelligent, answers questions slowly, speech slow and thick. Heavy upper lip. Walks slowly and unsteadily, shuffling gait. Tongue marked tremor, also hands. Glands, one behind s. mastoid muscle, otherwise not enlarged in neck. Both groin glands large and hard. Skin rough, pruritus.

*Circ. System.*—P. 80, reg. eq., vol. very small, tens. low, V. W. unthickened. *Heart*, a. b., not palp., u. b., 3rd r., r. b. 1½ inches, r. m. l., l. b. 4 inches to l. m. l. Ausc. Sounds very faint, no murmurs.

*Respir. System.*—R. 16, reg. eq. Cost. abdomen. Chest, increase in a. p. diameter. Note slightly hyper. resonant in front. Ordinary behind. Breath sounds faint. Vesic., no.; prolong. expiration.

*Aliment. System.*—Tongue large, flabby, very dirty, furred. Bowels constipated. *Abd.* Nil, inspect. Liver u. b., 5th and 6th, l. b., 1 inch below c. m. *Spleen*, u. b., 8th, l. b. 12th in mam. line at c. m. Not palp. Stomach not enlarged.

*Urinary Syst.*—No alb., bl., or sugar. No deposits.

*Lymph. Syst.*—Glands only one or two in post. triangle, none elsewhere in neck, enlarged in both groins. No venereal signs.

*Integ. Syst.*—Skin harsh, pruritus. No eruptions or old scars. Old small-pox.

*Nerv. System.*—Dull, apathetic look. Heavy, unintelligent. Speech slow. Sleep not marked. No cranial nerve paralysis.

*Motor Functions.*—No emaciation or wasting. Muscular nutrition good. Grip powerful, tremors marked in tongue and hands. Incoordination marked, *e.g.*, R.'s sign, cannot touch point of nose. Tremor increased in hands on putting cup up to lips.

*Sens. Functions.*—Pain sens. unimpaired, no hyperasthesia. Touch normal, tells accurately where touched. Temp. sense also good.

*Reflex Functions.*—Superficial present, ordinary. Knee reflexes absent. No clonus. Organic normal.

*Eye.*—Pupils eq., contracted, accom. to light very badly though slight react. during accommodation, no nystagmus or ocular paralysis. Conj. reflex acute.

*Hearing.*—Apparently normal, as also smell.

*Further Notes.*—August 23, 1902. No change.

September 2. Patient has lost flesh, tremor of tongue increased.

September 6. Change for the worse. Tremor increasing. Has come on quite suddenly.

September 8. Very much worse. Tremors very marked now all over body. Can hardly walk. Has to be supported. Emaciating rapidly.

September 17. Bedridden. Took to bed for good yesterday. Tremors increasing. Much worse. Passed motions in bed yesterday.

September 23. Completely bedridden. Tremors as before. Emaciation advancing rapidly. Some hyperæsthesia over neck and on tapping knees. Knee reflexes now present slight. Tremors of legs very marked.

September 24. Much the same.

September 25. A little better, still eats fairly.

September 27. Slight improvement, tremors very marked, however.

September 29. Better again. Crawled out to door yesterday, exceedingly feeble and tottering walk. Tremors excessive whole of body, shakes the bed. Hyperæsthesia when touched on neck, and resists being moved, this apparently causing pain.

October 1. In bed all day, tremors excessive, hyperæsthesia over trigeminal points.

October 5. About the same, has not been out of bed since October 1. Physical signs the same as on first. Purulent discharge from rg. ear. Knee reflexes gone.

October 6. Very ill and feeble, bedridden. Passing motions spontaneously. Still eats.

October 7. 10 A.M. About the same.

October 9. Very ill. Tremors excessive, shaking whole bed. Had a convulsive seizure at 6 P.M. Got rigid, no divergence of eyes, passed off in a few minutes.

October 10. No more seizures. Tremors very marked. Sordes on teeth and gums. Still a discharge from rg. ear. Very ill, still conscious, however.

October 14. Has remained about same. To-night very ill, in a state of semi-coma. L. p. done in evening.

October 16. About the same.

October 17. Very ill. Semi-comatose, breathing laboured.

October 18. Died 4 A.M.

## Table of Blood Counts.

1902.

| | | | | |
|---|---|---|---|---|
| September 17. | R., 3,720,000 ; | W., 8400 ; | Hb., 80 per cent. | Von Fleischl. |
| September 25. | R., 4,520,000 ; | W., 7800 ; | Hb., 86 per cent. | |
| September 26. | R., 4,000,000 ; | W., 6200 ; | Hb., 88 per cent. | |
| September 27. | R., 3,800,000 ; | W., 7000 ; | Hb., 84 per cent. | |
| September 30. | R., 3,480,000 ; | W., 12,500 ; | Hb., 80 per cent. | |
| October 1. | R., 3,200,000 ; | W., 12,000 ; | Hb., 80 per cent. | |
| October 2. | R., 3,400,000 ; | W., 10,600 ; | Hb., 80 per cent. | |
| October 3. | R., 3,480,000 ; | W., 7500 ; | Hb., 80 per cent. | |
| October 4. | R., 3,400,000 ; | W., 8100 ; | Hb., 81 per cent. | |
| October 5. | R., 3,100,000 ; | W., 6800 ; | Hb., 82 per cent. | |
| October 6. | R., 4,000,000 ; | W., 9300 ; | Hb., 82 per cent. | |
| October 7. | R., 4,000,000 ; | W., 6500 ; | Hb., 82 per cent. | |
| October 8. | R., 3,600,000 ; | W., 8700 ; | Hb., 80 per cent. | |
| October 9. | R., 3,120,000 ; | W., 9300 ; | Hb., 80 per cent. | |
| October 10. | R., 3,160,000 ; | W., 11,125 ; | Hb., 80 per cent. | |
| October 11. | R., 3,080,000 ; | W., 11,250 ; | Hb., 78 per cent. | |
| October 12. | R., 2,900,000 ; | W., 9700 ; | Hb., 76 per cent. | |
| October 13. | R., 2,800,000 ; | W., 6500 ; | Hb., 76 per cent. | |
| October 14. | R., 3,160,000 ; | W., 8125 ; | Hb., 78 per cent. | |
| October 15. | R., 2,800,000 ; | W., 21,800 ; | Hb., 76 per cent. ; | P. M. N., 72 per cent. ; L. M., 10 per cent. ; L., 16 per cent. ; E., 2 per cent. ; T., 0 per cent. |
| October 17. | R., 2,000,000 ; | W., 18,800 ; | Hb., 69 per cent. | |

*Post Mortem.* October 18, 10 A.M.

*External Appearances.*—Emaciated. Superficial bed sore over left trochanter.

*Chest.*—Heart. No pericarditis or pericardial effusion. Heart, muscle fibre pale, degenerated. Rg. ventricle ull of *post-mortem* clot. Valves all competent. Aorta healthy.

*Lungs.* No pleural adhesions, no fluid. *R.*, pale in colour, lower lobe slight congestion. *L.*, some slight congestion in lower lobe, otherwise healthy.

*Abdomen.*—No peritonitis. *Liver* enlarged, fatty and early cirrhosis. *Spleen* enlarged, slate-coloured capsule, old malaria pigment microscopically.

*Pancreas.*—Nil. *Intestines* not examined for parasites. Fæces showed ankylostoma and bilharzia ova only. *Stomach* slightly distended. *Bladder* normal. *Kidneys* normal.

*Lymph. System.*—En. glands neck, mesentery, etc., small and hard in consistence. *Neck.*—Nothing abnormal.

*Head.*—Calvarium unthickened. Dura not adherent. No excess cerebro-spinal fluid. Veins of pia arachnoid slightly congested. Sinus contained some fluid blood. No flattening of convolutions. Pia arachnoid stripped easily, no erosions. No excess subarachnoid fluid. Pia clear in consistence, no opacity. Lateral ventricles not dilated. Macroscopical changes practically then nil. Spinal cord nothing macroscopically. Rg. ear, from which pus came during life, showed no communication with interior of skull, proceeding from middle ear. Other parts of brain nothing macroscopically. Portions kept in Müller, alcohol and formalin.

*Histological Examination.*—Spleen, old pigment. Fæces, eggs of bilharzia and ankylostoma. Brain : Infiltration of meninges well marked. Vessels in interior surrounded by much cellular infiltration. Special methods : Decrease and atrophy of fibres in motor area. Some loss of Nissl's bodies in the cells. Spinal cord : Marchis stain shows a diffuse degenerative condition of the axis cylinders and sheathes of the nerves not specially limited to any area. Fat deposits in epineurium of the sciatic nerve, and some also in endoneurium.

CASE 13.—Sarumi. M., 16. Adm. August 28, 1902. Dis. September 29, 1902. Died. *P.M.* Native of Machogua (near Entebbe) :—

*Fam. Hist.*—Father and mother both dead, the patient cannot state of what disease. One brother alive and in good health.

*Pers. Hist.*—No previous illnesses. He was quite well up to two months ago, when he began to complain of headache and drowsiness—onset of illness was gradual—patient states that lately he has become much weaker.

*Present State.*—September 1. Patient looks drowsy ; unwillingness to respond to questions. He answers very slowly when spoken to. Well nourished. A few papules on arms, some pruritus. T. 99. R. 16. P. 110.

*Aliment. Syst.*—No subject sympt. Tongue furred, fine tremor. Nothing abnormal at the examination of the abdomen. Liver, spleen, not enlarged, no intestinal symptoms. *Micr. exam. of fæces* : many ova of bilharzia.

*Circ. System.*—No subj. symptoms. Pulse of reg. rhythm, equal, low tension. Apex beat impalp., areas of relative and absolute dulness have normal limits. Ausc. nothing abnormal.

*Blood.*—Glands enlarged in both sides neck, axilla, groin.

*Lymph. System.*—

*Respir. System.*—Resp. cost. abd. regular. Inspec. palp. perc. ausc. of chest, nothing abnormal.

*Urinary System.*—Exam. of urine. No alb., bl., or sugar. Sp. gr. 1016.

*Nervous Syst.*—Patient looks drowsy, unwilling to respond to question. No abnormalities of speech. No headache, no giddiness. Cranial nerve functions normal. No paralysis or contractures, no tremor of hands, very slight tremor of tongue. Romberg's symptom absent. Sensory functions normal. Superf. refl. normal. Knee jerk increased both sides. Org. refl. normal.

*Special Senses. Eye.*—No ocular paralysis, no nyst., pupils eq., ready reaction to light and accom.

*Further Notes.*—September 8. No change.

September 23. Very drowsy. Fine tremor in tongue, and now in hands. Stays a good deal in bed.

September 27. Drowsiness much more marked, and general state much worse. Tremor increased. Leaves bed very little now. Passed urine in bed for first time yesterday. Pulse just perceptible at wrist. Tens. very low.

September 28. Much worse. Very cold, with sub-normal temperature. P. imperceptible at wrist. Passing motions in bed. Semi-comatose, will not respond to questions. Mucus welling from mouth. 6 P.M. Pulse very slow, 38. In a state of coma; cold extremities. Mucus welling from mouth.

September 29. Died 8.30 A.M. in a state of coma. No fits.

## Table of Blood Counts.

| 1902. | | | |
|---|---|---|---|
| September 18. | R., 4,200,000; | W., 6800; | Hb., 80 per cent. |
| September 28. | R., 4,540,000; | W., 8100; | Hb., 87 per cent. |
| September 29. | R., 4,480,000; | W., 19,375: | Hb., 86 per cent. |

*Post Mortem Examination. September* 29, 9.30 A.M:—

*Ext. Appearances.*—Body well nourished, not emaciated. Skin rough and scaly over tibia, otherwise smooth. No old scratch marks, and no eruptions or old scars on trunk. Two old white scars on penis just on skin above glands. Skin around somewhat thickened.

*Thorax.*—Pericardium, no fluid. Heart, large dilation of rg. ventricle. Left not dilated. Rg. auricle full of *post-mortem* clot extending into rg. ventricle. Valves all competent and healthy. Aorta healthy. No pleural effusion, no old adhesions.

*Lungs.*—Healthy. *R.* Some œdema of lower lobe, no congestion, bronchitis, or pneumonia. *L.* Nothing abnormal. Bronchial glands enlarged about size of pea.

*Abdomen.*—No peritonitis or fluid in peritoneum. *Liver* enlarged, pigmented capsule, section pigmentation, and some increase in the fibrous tissue around the lobules. No congestion. *Gall Bladder* contained a little dark-coloured bile. *Spleen* enlarged, pig. capsule, slate-coloured section. Pigmented old malaria.

*Kidneys.*—*R.* Capsule strips easily. Section normal. *L.* Ditto to. rg.

*Pancreas.*—Nil.

*Stomach.*—Slightly dilated, contained some slimy mucus.

*Duodenum.*—Some congested points, two small mucous polypi.

*Jejenum and Ileum.*—Lower part some fine congestion.

*Cæcum.*—Some dark fæces. No ulceration anywhere.

*Parasites.*—Trichocephalus dispar many, no ankylostomes or ascaris.

*Bladder.*—Nil.

*Neck.*—Nothing abnormal. Larynx, vessels and fauces all normal.

*Lymph. System.*—General enlargement of glands, neck small, size of small almonds. Those in groin and femoral region the same size. Mesenteric slightly larger, and on post. wall of abdomen behind pancreas two very large glands the size of a large walnut. Section hard, no suppuration. Bronchial glands size of peas. Epitrochlear about size of small almonds.

*Head.*—Calvarium rather thin. Dura mater not adherent to calvarium, slight excess of sub-dural fluid. Veins over vertex extraordinarily congested, turbid, and distended with dark-coloured blood. Smaller capillaries also congested. Pia arachnoid transparent, strips easily without erosion. Sub-pial fluid very slightly in excess. A fair quantity of fluid in lateral ventricles. Apart from the congestion of the vessels very little macroscopic change. Base similar. Nothing macroscopic in interior pons, cerebellum or medulla. Ventricles not dilated.

*Spinal Cord.* Dura healthy. Some congestion of vessels underneath. Section nothing apparent macroscopically.

*Histological Examination.*—Spleen and liver recent malarial pigment. Exam. of fæces. No ova ankylostomes, ascaris. Many ova of Bilharzia. Never blood or ova in urine, so a case of Bilharzial infection of rectal veins.

Case 14.—Semsoni. M. 24. Ad. September 5, 1902. Dis. October 3, 1902. Died. *P.M.* Native of Buse :—

*Fam. Hist.*—F. d. fever. M. d.? B. 0. S. 0. Ch. 0.

*Pers. Hist.*—Labourer. Food, bananas, has lived in poor circumstances.

*Previous Illnesses.*—Has suffered from fever.

*State on Admission.*—T. 100. P. 96. Heavy dull appearance. Answers questions very slowly. Walk slightly impaired. Very slight tremor hands and tongue. Headache occipital and pain in left side of neck. No pains in chest. Skin slightly rough, no pruritus or eruptions. En. rather large glands in p. triangles, smaller in ant. triangles and submaxillary, also palpable in axillæ, groins and epitrochlear. Not emaciated.

*Circ. System.*—P. 108, reg. eq., tens. very low. Vol. very small. V. W. unthickened.

*Heart.*—Epigast. puls. A. B. not palp., u. b. 3rd rib, l. b. 4 inches to l. of m. l., r. b. $\frac{1}{4}$-inch to rg. of m. l. *Ausc.* Sounds rather indistinct at base. Clear at apex, no murmuring.

*Resp. System.*—R. 20, cost. abd. Chest well formed. Percus. no dulness. Note reson. all over. *Ausc.* Vesicular ordinary.

*Aliment. Syst.*—Tongue large flabby. Fur on dorsum. Some small pigmented spots at sides. Fæces nil. Appetite good. Bowels constipated. *Liver* u. b. 5th and 6th, l. b. 1 inch below c. m. *Spleen* u. b. 8th, l. b. 12th, in mam. line at c. m. just palpable. *Stomach* not enlarged.

*Urinary System.*—Sp. gr. 1018. No alb., bl., or sugar. No deposit.

*Lymph. System.*—Glands large in size (almond) in post. triangles, also enlarged glands in all the other areas.

*Integ. System.*—Old scar on rg. side of neck over sterno mastoid. Skin a little rough, no pruritus or eruptions.

*Nervous System.*—Dull, heavy and very stupid. Speech slow and impaired. Drowsy appearance, but does not actually sleep. Headache in back of head. No cranial nerve paralysis. Tremor tongue and arms.

*Motor Functions.*—No emaciation or wasting. Musc. nutrition good. Grip fair. Fine tremor in tongue and hands. Incoordination distinct. R.'s symptom. Cannot touch tip of nose accurately.

*Sensory Functions.*—No impairment.

*Reflex Functions.*—Superficial present well marked. Knee reflexes brisk, eq. on both sides. No clonus. Organic normal.

*Special Senses.*—*Eye.* Conj. anæmic. Trace of jaundice. Pupils small, equal, react poorly to light. Well to accommodation. No nystagmus or ocular paralysis. *Hearing* apparently unimpaired.

*Further Notes.*—September 10. Worse, drowsiness increasing. Walk distinctly impaired, shuffling in character. Tremors slightly increased.

September 23. Gradually getting worse.

September 24. Tremors increased. Very dull and apathetic. Stays mostly in bed. Walk worse.

September 27. Much worse. Passing water in bed now. Does not get up. Tremor tongue, arms and legs very marked.

September 28. Worse. Emaciation progressing rapidly. Contracture of limbs marked, *e.g.*, legs on thighs, latter on abdomen. Tremors of tongue and limbs worse, knee reflexes gone. Small bed sore on rg. trochanter. Hyperasthesia on being touched on neck or legs, cries out. Rapidly getting worse.

September 29. Worse. Extremities cold. Shrinks greatly when one makes even a pretence of touching him. Does not respond to questions. Not actually sleeping, but approaching coma.

October 1. Very ill, still distinctly conscious. Tremors in tongue not so bad. Contracture of limbs increasing. Very marked hyperasthesia over points of exit of 5th nerve, also shrinks when touched elsewhere. Two small excoriations appeared over front of chest. Passing motions spontaneously. Emaciation marked.

October 2. About the same.

October 3. 11 A.M. Very bad, eyes closed. Breathing deep and laboured. L.P. done 12 noon. Pressure great, fluid gushed out. Breathing became ordinary immediately after. 5 P.M. Breathing deep and laboured again, rapid pulse impercept. Died 6 P.M. in a state of semi-coma.

## Table of Blood Counts.

| 1902 | |
|---|---|
| September 11. | R., 2,771,000; W., 9000; Hb., 65 per cent.; P. M. N., 50 per cent.; L. M., 23 per cent.; l., 20 per cent.; T., 5 per cent.; E., 2 per cent. |
| September 28. | R., 2,260,000; W., 17,500; Hb., 63 per cent. |
| September 29. | R., 2,960,000; W., 16,800; Hb., 64 per cent.; P. M. N., 74 per cent.; L. M., 6 per cent.; l., 16 per cent.; E., 2 per cent.; T., 2 per cent. |
| September 30. | R., 3,300,000; W., 16,800; Hb., 65 per cent. |
| October 1. | R., 2,500,000; W., 18,750; Hb., 60 per cent. |
| October 2. | R., 2,600,000; W., 12,600; Hb., 60 per cent. |
| October 3. | R., 3,400,000; W., 19,300; Hb., 65 per cent. |

*Post Mortem, October* 4. 6 A.M.

*Ext. Appearances.*—Body much emaciated. Bed sore on rg. trochanter. Two excoriations, one over sternum, and one to the left. Old scar rg. side of neck over sterno mastoid. Skin a little rough, no eruptions or old scratch marks.

*Chest.*—No pericardial fluid.

*Heart.*—R. ventricle dilated, left ordinary. Muscular substance pale, slightly degenerated. Valves competent. Aorta healthy. Rg. auricle full of ante mortem clot extending into rg. ventricle.

*Lungs.*—No fluid in pleura. *R.* Pale, almost white colour. Œdema and some congestion at base of lower lobe. *L.* Pale and bloodless, crepitant, some œdema of lower lobe, no congestion or pneumonia.

*Abdomen.*—Exam. of fæces, before death, Bilharzia and other ova. No peritoneal fluid or adhesions. *Liver.* Enlarged, pigmented old malaria. *Spleen.* Enlarged, not markedly so. Capsule slate-coloured. Section reddish-brown. Old malaria.

*Kidneys.*—*R.* Very pale, capsule strips readily. Superficial capillaries infected, as also those in interior. No inflam. changes. *L.* Ditto to right.

*Pancreas.*—Nil.

*Intestines.*—Normal. Intestinal parasites. Ankylostoma, plenty, in duodenum scanty, in upper part of jejunum many, and extending down it for a considerable distance. Ascaris few. Trichocephalus few.

*Rectum.*—One small patch, blood-stained, near anus.

*Bladder.*—Healthy.

*Lymphatic Syst.*—Mesenteric glands size of small peas, also those in neck and other areas. No large glands on post. Abdom. wall enlargement slight therefore.

*Neck.*—Nothing abnormal.

*Head.*—Calvarium thin. Dura not adherent. Fluid not now in excess after the L. puncture of yesterday. Distinct flattening of convolutions on vertex. Vessels over vertex and other surfaces of brain full of dark blood congested, also those over cerebellum. Capillaries also slightly congested. Pia arachnoid clear in consistence (slight excess of fluid), colour slightly turbid. Strips easily without erosion. Lat. ventricles distended fluid in excess, very slight turbidity. No other macroscopic change in other parts of brain. Presented as a whole general features of Sleeping Sickness. Congestion of vessels, otherwise macroscopically, nothing.

*Histological Examination.*—Spleen and Liver, old malarial pigment.

*Brain.*—Presents a well marked meningeal exudation more marked than usual. Infiltration round vessels also well marked. *Pons* and *Medulla*, well marked infiltration round vessels in substance. *Cord.*—Many vessels show the usual exudation around. Meningeal surf., well marked infiltration. *Heart Muscle.*—Markedly degenerated in substance, also several areas of leucocytic infiltration especially round the vessels. Skeletal muscle gastrocnemius, degenerated fibres not clear and separated from each other.

Case 15.—Alfonse. 15 M. Ad. Sept. 8, 1902. Still in hospital. Native of Kesube:—

*Fam. Hist.*—F. l. M. l. h. B. l. h. S. 0.

*Pres. Hist.*—At R. C. Mission, did work as a boy of light nature.

*Prev. Ill.*—Has had colds and some fever.

*State on Ad.*—Heavy drowsy look, heavy lips, trace of jaundice in conjunctiva. Very faint tremor in tongue, none in arms. Skin dry and harsh. No eruptions or pruritus. A large mass of glands in left post. triangular, smaller glands in other p. triangular and in ant. and sub-maxillary. Epitroch. not palp. axillary and groin slight. Speech slow. Walk impaired, slight shuffling gait.

*Circ. System.*—P. 100. Reg. equal, Vol. mod., tens, mod. V. W. unthickened.

*Heart.*—A. B. 4th sp. 3 inches from m. l. u. b. 3rd rib, l. b. 4 inches from m. l. R. C. $\frac{1}{4}$ inch to rg. of m. l. *Ausc.* Sounds clear and distinct, no murmurs.

*Resp. System.*—No cough. No sputum. R. 16. Reg. equal., cost. abdomen. Chest well formed. *Perc.* Reson. note all over. Ausc. breathing vesic., no prolongation of expiration.

*Aliment. System.*—Tongue, large, flabby, slight fur. Teeth good, tonsils not enlarged. Appetite good, bowels constipated. Abd. shows nothing on inspection. *Liver*, u. b. 5th and 6th l. b. c. m. not palp. *Spleen*, u. b. 7th, l. b. 12th at c. m. in mam. line not palp.

*Urinary System.* No albumin, blood, or sugar. No deposit.

*Lymphatic System.*—Many enlarged glands (one size of large almond on left side) in both p. triangles. A few slight in Ant. triangles. None sub-max., or sub-mental, or in groins. Epitroch and axillary not palp.

*Integ. System.*—Skin lost its gloss. No pruritus or eruptions, dry.

*Nervous System.*—Heavy drowsy look with a frightened aspect. Speech fairly quick, does not sleep much. No cranial nerves paralysis.

*Motor Functions.*—No emaciation or wasting. Muscular nutrition good. Faint tremor in tongue, grip good. No incoordination.

*Sens. Functions.*—No impairment.

*Reflex Functions.*—Superficial, all present, brisk. Knee not exaggerated, about ordinary, equal on both sides. Organic normal. No clonus. *Eye.* Pupils equal. mod. dilated, react to light and accommodation. No nystagmus. Hearing and smell unimpaired.

*Further Notes.*—September 23. Very little change. Tremor in tongue slight. Still heavy expression.

September 25. Seized with acute pain in abd., bowels constipated. Pressure over region of stomach causes patient to cry out with pain. Hot fomentations applied.

September 26. Pain in abdomen gone—better.

September 29. Drowsy, but does not show much change.

October 5. In *statu quo.*

October 10. About the same, brighter if anything.

October 20. About the same.

October 28. Appears a little better. No drowsiness. Goes about out of doors all day.

In conclusion, we have to tender our best thanks to Colonel Sadler, His Majesty's Commissioner of Uganda; to Dr. Moffat, C.M.G., Principal Medical Officer of Uganda; to Drs. Bagshawe, Hodges, Wiggins and Sly, for their kindness and help in forwarding our work in every possible way, and for much useful information and material.

APPENDIX.

---

# *FILARIA PERSTANS* AND ITS RELATIONSHIP TO SLEEPING SICKNESS.

By GEORGE C. LOW, M.A., M.B., C.M.

Following the instructions issued by the Royal Society, namely, that Sleeping Sickness might be a disease due to the nematode parasite *Filaria perstans*, I took up this part of the work specially, and finally decided that there was no connection between the two.

This hypothesis was first advanced by Sir Patrick Manson, who demonstrated the presence of this parasite in three cases of this disease which were brought to England, one under the care of Sir Stephen Mackenzie at the London Hospital, and two at Charing Cross Hospital. Having just returned from the West Indies and British Guiana, where I had been making a special study of the different forms of *Filaria*, I had the advantage of possessing a vast amount of material bearing directly on this question before I started for Africa.

On arriving in Uganda, the method adopted to determine whether *Filaria perstans* could be incriminated in the production of Sleeping Sickness was to examine thousands of individuals' blood in different areas and to see if the parasite gave rise to any pathological changes. The special procedure was: (*a*) the examination of sick and sound in areas where Sleeping Sickness was rife; (*b*) the examination of people in areas where Sleeping Sickness had not appeared or had never been known; and (*c*) pathological data.

## (A) *Areas with Sleeping Sickness.*

1. *Sese.*—This, a group of islands in the lake, was chosen on account of its convenience, and on account of the large amount of the disease present there. An examination of sixty-eight such cases showed that 66 or 97 per cent. had *Filaria perstans*, but against this, on the other hand, eighty-six individuals out of a hundred, carefully selected as free from Sleeping Sickness, also exhibited the same parasite in their blood.

2. *Entebbe and Neighbourhood.* An examination of fifty well-marked cases of Sleeping Sickness showed 45 or 90 per cent. with *Filaria*

*perstans* in their blood, whereas 100 ordinary individuals only showed *Filaria* in fifty-two, or 52 per cent.

In both these instances it is at once seen that there is a greater percentage of *Filaria* in the Sleeping Sickness cases than in the ordinary apparently healthy individuals. It is quite conceivable, however, that persons in the marasmic condition of this complaint may more easily become infected with parasites than those in health, and it must further be stated that in this latter estimation five of the Sleeping Sickness cases did not show *Filariæ* in their blood on the first examination, and then only one or two in 5 c.mm. of blood on further examinations. Of the 100 healthy individuals only one examination of 5 c.mm. of blood from each was possible.

3. *Kavirondo and the Kavirondo Islands.*—Dr. Hodges had already shown, before the commission arrived in Uganda, that, though Sleeping Sickness was very prevalent in the Kavirondo Islands, *Filaria perstans* on the other hand was very rare. In Nyala only three out of thirty-four cases of the disease showed the parasite, and in normal individuals only one out of fifty. Again, in Sigula Island, out of forty-five cases only three exhibited the parasite, and keeping up the contrast only two out of fifty-two healthy cases. Still more striking were the figures of Dr. Wiggins, of Kisumu, who found that not one out of 150 Wasemi cases of the disease had *Filaria perstans* in their blood, the same condition applying to the general healthy Kavirondo population of Kisumu, a place situated at the extreme east of the lake. A special visit to this place on my way home to England confirmed Dr. Wiggins' report as to there being no *Filariæ* there, yet a census, which Mr. Hobley, the Sub-Commissioner, kindly made for me, showed that there were seventy cases of Sleeping Sickness within 2 miles of his house at Kisumu (October, 1902). The disease at that time was spreading amongst the Kavirondo with great rapidity, and large numbers had died of it.

## (B) *Areas Without Sleeping Sickness.*

1. *British Guiana.*—A few assertions, not supported by any evidence whatsoever, have been made that Sleeping Sickness has been seen in British Guiana amongst the Buck Indians. It has been said that the Indians when they fall sick take to their hammocks and lie there till they die. That they should do so is not by any means wonderful, as their hammocks are their beds, but this does not prove in the least that they are suffering from Sleeping Sickness. While travelling in the forests of the interior of British Guiana, I specially looked out for this disease, but though I often saw sick Indians lying in hammocks, I found that they were generally suffering from malaria and other disease and never from Sleeping Sickness. Mr. Perkins, Acting Commissioner of Gold Mines, who has journeyed extensively through

the forests of the interior, informed me that the Indians were unaware of such a disease, and he personally had never heard of it. Again, though the Medical Staff in Georgetown, the capital of British Guiana, have been on the outlook for this malady for the last six years, no case has even been seen. The disease, therefore, I am convinced, does not exist in British Guiana. An examination of the blood of these aboriginal Indians, however, reveals the fact, already pointed out by Daniels and Ozzard, that *Filaria perstans* is exceedingly common, no fewer than 50 per cent. of them having it, some with it alone, while others very often have a sharp-tailed variety also, a different species. These nematodes seem to give rise to no symptoms.

2. *Unyoro.*—An examination of 100 individuals' blood from this large district near the Albert Lake, made by Dr. Moffat, C.M.G., and myself, gave seventy-four infected with *Filaria perstans.* As in British Guiana, Sleeping Sickness has never been known there.

3. *The Upper Nile Districts.*—Sleeping Sickness is unknown in all the Upper Nile stations. The percentage of filariasis is much lower here, for example 8·4 per cent. in the Alurs from Wadelai, but still it exists.

## *Conclusions.*

The results of these examinations show :—

1. That there are areas where Sleeping Sickness exists and *Filaria perstans* is absent.

2. That there are areas where *Filaria perstans* is very common and yet there is no Sleeping Sickness.

3. That there are areas where both Sleeping Sickness and *Filaria perstans* are found, the parasite existing, however, in the blood of the healthy as well as in that of the sick.

Double infections of the blunt and sharp-tailed nematodes, *Filaria perstans* and *Filaria demarquaii*, are very commonly met with in British Guiana, but single infections of either may occur. Out of 163 aboriginal Indians examined at one place, there were thirty-eight with double infections, fifty-six with *Filaria perstans* alone and eleven with *Filaria demarquaii* alone. The average number affected with *Filaria perstans* is from fifty to sixty per cent. (C) pathological data which render it unlikely that *Filaria perstans* is the cause of Sleeping Sickness.

The occurrence of *Filaria perstans* in such large numbers of healthy people, both in areas with Sleeping Sickness and in those without, show that this worm probably produces no pathological symptoms whatever. It has been contended that because *Filaria nocturna* does not always cause pathological symptoms, that the same may be the case with *Filaria perstans.* One has only to look for a moment at the sites of selection in the human body of those two nematodes to see

## Table showing the Prevalence of *Filaria perstans* in some Areas.

### AFRICA.

| No. | Race. | District. | No. of healthy individuals examined. | Percentage with *F. perstans*. | Percentage with *F. nocturna*. | Presence or absence of Sleeping Sickness. |
|---|---|---|---|---|---|---|
| 1 | Baganda | Entebbe | 100 | 51 | Not determined | Very prevalent. |
| 2 | „ | „ | 100 | 42·2 | 2·2 | „ |
| 3 | „ (children under ten) | „ | 100 | 14·2 | 0 | „ |
| 4 | Baganda | Bulemezi | 100 | 62 | Not determined | A few cases. |
| 5 | „ | Ndeji Bulemezi | 100 | 27 | „ | A few suspicious cases (Mr. Leaky). |
| 6 | „ | Kago | 100 | 62 | „ | A few cases (Cook). |
| 7 | „ | Mugema | 100 | 50 | „ | ? |
| 8 | „ | Buddu | 100 | 20 | „ | A few cases reported. |
| 9 | „ | South Buddu | 100 | 30 | „ | Absent (Bagshawe). |
| 10 | „ | Gimbo Chagwe (examined by Moffat) | 100 | 90 | „ | Very prevalent (Moffat). |
| 11 | Wasese | Sese | 100 | 86 | „ | „ |
| 12 | Bavuma | Buvuma Island (off coast of Usoga) | 50 | 78 | „ | „ |
| 13 | „ | Bugaia Island | 50 | 2 | „ | Prevalent. |
| 14 | Kavirondo | Kisumu | 30 | 0 | „ | Very prevalent. |
| 15 | „ | „ (examined by Dr. Wiggins) | 100 | 0 | „ | „ |
| 16 | Wasemi | Nr. Kisumi (examined by Dr. Wiggins) | 150 | 0 | „ | „ |
| 17 | ? | Ankole (examined by Moffat and self) | 140 | 7·8 | „ | Not known. |
| 18 | Wanyora | Unyoro „ „ | 100 | 74 | „ | „ |
| 19 | Alur | Wadelai (examined by Bagshawe and self) | 50 | 8·4 | „ | „ |
| 20 | Nubians | Nile districts | 100 | 6 | 2 | „ |
| 21 | Swahili | Coast | 100 | 2 | 26 | „ |
| 22 | Europeans | Entebbe, &c. | 20 | 0 | Not determined | Very prevalent. |

Table showing the Prevalence of *Filaria perstans* in some Areas—*contd.*

BRITISH GUIANA.

| No. | Race. | District. | No. of individuals examined. | Percentage with *F. perstans* or *F. demarquaii.* | Percentage with *F. nocturna.* | Presence or absence of Sleeping Sickness. |
|---|---|---|---|---|---|---|
| 23 | Carib and Arawak Indians | Pomeroon River, British Guiana...... | 100 | 71 | Not determined | Absent. |
| 24 | Carib Indians .... | Demerara River.................. | 10 | 60 | ,, | ,, |
| 25 | Warau Indians ... | Waini River....................... | 30 | 60 | ,, | ,, |
| 26 | Akawoio Indians.. | Barima River...................... | 20 | 36·6 | ,, | ,, |

that this does not follow at all. The adult or parental forms of *Filaria nocturna* live in the lymphatics, and the changes they give rise to are purely mechanical. Anything may at any time cause the death of the parasites, or a collection of a number may block the tube in which they lie, and consequent changes involving the lymphatic system are then apt to set up. In the case of *Filaria perstans* it is quite different; the adults here are living in the connective tissues of the mesentery, and all that could happen by their death would be perhaps a little local inflammation, though it is improbable that even this would take place.

The only other hypothesis would be (1) an accumulation of embryos in the fluids of the brain, which does not occur, or (2) the production of a toxin from the adult or embryo, an extremely improbable theory, as none of the other *Filariæ* are known to give rise to such products. *Filaria demarquaii*, which resembles *Filaria perstans* very closely in every respect, even to the site selected by the adults, gives rise to no pathological symptoms, and it is difficult to see by analogy why *Filaria perstans* should.

The final conclusions then one comes to, both from the distribution and the pathological anatomy, are that *Filaria perstans* has nothing to do with Sleeping Sickness, its presence in these cases being only a coincidence, as is the presence of *Ankylostoma duodenale*, *Ascaris lumbricoides*, *Trichocephalus dispar*, and *Bilharzia hæmatobia*.

C. Christy.

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J. L. Todd

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By CUTHBERT CHRISTY, B.M., C.M. (Edin.).

(Written July 14, 1903,—Received (with revisions), Sept. 5, 1903.)

(With 4 plates and 5 maps.)

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### PLATES.

1. In the Shambas, Buvuma Island. Showing a hut, open round the edges—a common form in this island—and how closely it is surrounded by bananas.
2. Sleeping Sickness and Starvation, Buvuma Island. Showing the condition of the remnants of the famine-stricken inhabitants. In the foreground to the left is a very well-marked case of the bloated type of Sleeping Sickness, in a woman. Other cases in various stages are seen in the group.
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4. The Last Stage—Lethargy. In the hut from which this man was carried, without waking, was another man in a similar condition; and a woman, who, after much inducement, was just able to get up and sit outside.

## 1. EARLY RECORDS OF SLEEPING SICKNESS.

The first descriptive account of the disease would appear to be the outcome of the observations of Dr. T. Winterbottom, who, in 1803, records the following* :—

> "The Africans are very subject to a species of lethargy, which they are much afraid of, as it proves fatal in every instance. The Timmanees call it márree, or 'nluoi, and the Bulloms nagónlôe, or kadura; it is called by the Soosoos, Kee Kóllee Kondee, or sleepy sickness, and by the Mandingoes seenoyuncaree, a word of similar import. This disease is very frequent in the Foo-la country, and it is said to be much more common in the interior parts of the country than upon the sea coast. Children are very rarely, or never, affected by this complaint, nor is it more common among slaves than among free people, though it is asserted that the slaves from Bennin are very subject to it. At the commencement of the disease, the patient has commonly a ravenous appetite, eating twice the quantity of food he was accustomed to take when in health, and becoming very fat. When the disease

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* 'An Account of the Native Africans in the Neighbourhood of Sierra Leone,' by Thomas Winterbottom, M.D., London, 1803, vol. 2.

has continued some time, the appetite declines, and the patient gradually wastes away. Squinting occurs sometimes, though very seldom, in this disease, and in some rare instances the patient is carried off in convulsions. Small glandular tumours are sometimes observed in the neck a little before the commencement of this complaint, though probably depending rather upon accidental circumstances than upon the disease itself. Slave traders, however, appear to consider these tumours as a symptom, indicating a disposition to lethargy, and they either never buy such slaves, or get quit of them as soon as they observe any such appearances. The disposition to sleep is so strong as scarcely to leave a sufficient respite for taking of food; even the repeated application of a whip, a remedy which has been frequently used, is hardly sufficient to keep the poor wretch awake. The repeated application of blisters and of setons has been employed by European surgeons without avail, as the disease, under every mode of treatment, usually proves fatal within three or four months. The natives are totally at a loss to what cause this complaint ought to be attributed. Sweating is the only means they make use of, or from which they hope for any success; this is never tried but in incipient cases, for when the disease has been of any continuance they think it in vain to make the attempt. The root of grass, called by the Soosoos, kallee, and the dried leaves of a plant called in Soosoo, fingka, are boiled for some time in water, in an iron pot; when this is removed from the fire the patient is seated over it, and is covered over with cotton clothes, a process which never fails to excite a copious perspiration. This mode of cure is repeated two or three times a day, and is persisted in for a considerable length of time, until the disease is arrested, or appears to be gaining. No internal medicines are given in the complaint."

Dr. P. A. Guérin, in a thesis on Sleeping Sickness presented to the Paris University in 1869, says, referring to the immigration of Africans into Martinique, in the West Indies—

> "For about 12 years, during a period when the immigration of Africans into Martinique was considerable, we were called upon to treat Sleeping Sickness on the various landed estates, as well as in the civil hospital at Fort de France, in the discharge of our duties."

Guérin selects several from 148 cases treated by him during his stay in the island, and gives full clinical and other data, followed by the results of autopsies. His first case is that of—

(1) Félise, whose age was about 25, and who had been in Martinique for 5 years. He supplies the results of his daily

observations, and notes especially the various attacks of somnolency, which gradually increase, so as to leave ultimately but few intervals in which the patient remains awake, till January 22, when gradual improvement begins, and continues till March 10, when he is declared fit to resume his work. On September 2, however, 7 months after leaving hospital, he is again admitted with similar symptoms, grows gradually worse, and on October 25, spasms appear, then stupor again, and finally, November 2, without awakening he dies. The period of incubation is apparently 5 years or more.

(2) Loubendeau, brought to hospital on May 11, 1859, three days after arriving in Martinique. The physician on board had noticed his drowsiness a few days after leaving the Congo. The patient died on November 6.

(3) Joseph, came to hospital of Fort de France on July 15, 1865. Is 19 years of age, and had arrived in the Colony 5 years previously. He died on October 25. The incubation period again appears to be 5 years, or over.

(4) Mathieu, a Creole, age 15, resident in Colony 3 years. Towards November, 1863, "seemed to become sad, his manners lazy, his walk and all his motions slow," followed by increasing periods of somnolency till death on June 22, 1864.

(5) Nagou, a woman, age 17, resident 4 years, admitted May 5, died September 10.

On reading, however, a summary of Dr. Guérin's thesis, published in the 'Archives de Médecine,'* one is led to doubt the diagnosis of many of his cases. So large a number as 148 in Martinique alone during the comparatively short period of 12 years is in itself startling.

In referring to the fact that the cases of Sleeping Sickness treated in Martinique occurred only among negroes who had come to the island from the Congo district, Guérin says†—

> "Not all black-skinned people are equally liable to contract 'hypnosie.' The black Creoles of the Antilles, for instance, have been employed without bad consequences in the factories of the firm Régis, and have never caught it. It broke out, however, with great virulence among the Africans who had come over during the last emigration of blacks from the Congo into the Antilles. Sleeping Sickness has frequently appeared among them, long after their leaving their homes. This peculiarity of affecting only a special group of individuals allows of the assumption that there exists a special predisposition toward the disease among the negroes from the Congo. Nevertheless, the older negroes from

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* 'Archives de Médecine,' VIe Série, vol. 14, Paris, 1869.

† Thesis.

the Congo who were landed there in agreement with the treaty, and who have been in the Colony some 40 years, have not a single case to show."

Careful search that I caused to be made among books of travel referring to the West Coast of Africa from the beginning of the 16th century, has failed to bring to light records of the disease anterior to Dr. Winterbottom's account. Dr. Corré, who investigated the literature of the subject, met with no better success, for in 1876 he writes*—

> "Before undertaking my journey into the region of Nelvane, I went through all the books where I hoped to find some information regarding the disease. My researches had a negative result. Labat, who wrote his 'Nouvelle relation de la côte occidentale d'Afrique' on the basis of comprehensive and very well-selected material, and Adanson, who visited Portudal towards the middle of the 18th century, mention nothing that refers to Sleeping Sickness. The books referring to the treatment of the slaves, so rich in horrible details, enumerate the various causes of their mortality, and their diseases observed; some of them speak, it is true, of languor, exhaustion, debilitating causes, physical as well as moral, but nowhere, even in passages quoted from medical reports, is there any mention of somnolency. Nor have books, written in America during the period of slavery, given me any information."

## 2. Geographical Distribution.

Dr. H. Rey in an article entitled "Maladie du Sommeil" contributed to the 'Dictionnaire de Médecine et de Chirurgie pratiques,' vol. xxxiii (1882), summarises what was known of the geographical distribution of the disease at that date as follows:—

> "Sleeping Sickness has been observed on the whole west coast of Africa from the Portuguese Possessions in the south (Loanda, Benguela), up to the mouth of the Senegal; also on the islands of the Gulf of Guinea (Princes Island, Saint Thomas, Fernando-Po); and finally on the French Antilles among negroes who had come from Africa. At some points the disease occurs with exceptional frequency, as for instance, in Senegambia in the districts of Joal and Portudal; the village of Nianing in the latter is pointed out by Corré as a focus of the malady."

Until 1901 this remained the general distribution of Sleeping Sickness, with the addition that the disease had appeared as far up the

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* A. Corré, "Contribution à l'étude de la maladie du sommeil," 'Gaz. Méd. de Paris,' 1876.

Congo as Stanley Falls; also in 1898 at Jebba on the Upper Niger, Dr. D. Rees had a well-marked case under his care from the Royal Niger Company's Constabulary; and subsequently I had two more cases, one a servant from the coast, the other a Yoruba in the West African Frontier Force, who was in my charge in the Field Force Hospital at the same station.

### 3. THE OUTBREAK IN UGANDA.

As already seen, prior to 1901, Sleeping Sickness had only been recorded from Western Africa, excepting those cases imported into the West Indies from the Congo. In the early part of that year, reports were received by the Foreign Office from H.M. Commissioner in Uganda, with reference to the apparently widespread epidemic of the disease in the Protectorate.

The first cases were admitted in April, 1901, at the Church Missionary Society's Hospital at Mengo, in the Province of Uganda, an institution that had been in existence for about five years, under the charge of Dr. A. R. Cook and Dr. J. H. Cook. These officers had never met with the disease previously, nor was it known apparently to the natives.

Of the first eight cases, four came from Busoga,* and the natives affirmed that the disease, called by them "Mgota," was much more prevalent in that province than in the Kingdom of Uganda. Later in the year, the cases became so frequent that the missionaries were obliged to refuse them admission to their hospital, and on this being reported to Entebbe, the administrative headquarters of the Uganda Protectorate, Dr. Aubrey Hodges, Medical Officer for Busoga, was instructed to make an investigation, and report upon the disease throughout his district. Towards this end, he made a canoe journey to "the most important places along the north coast of Lake Victoria from Jinja to Nyala, and the chief Busoga, Buvuma, and Kavirondo Islands."† He found that the disease was "most prevalent from Lubas to the borders of Kavirondo, and in Buvuma and the Busoga Islands."‡ In his district alone, 20,000 persons were reported to have already succumbed.

### 4. THE FORMATION OF A COMMISSION OF INVESTIGATION —INSTRUCTIONS RECEIVED.

As the result of the above reports, and at the instigation of the Foreign Office, a Commission was appointed by the Royal Society to proceed to Uganda for the purpose of investigating the cause of the disease, and as a member of that Commission, I received from the Society in the first week of June, 1902, the following amongst other instructions, under the heading of "Suggestions to Observers":—

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* 'Journal of Tropical Medicine,' July 15, 1901.

† 'Report on Sleeping Sickness,' by Dr. Hodges, 1902.

‡ *Ibid.*

> "Dr. Christy [is] to proceed round [the] north of Lake [Victoria] carefully collecting and examining bloods so as to ascertain where the area of the presence of *Filaria perstans* commences, and where that of Sleeping Sickness commences."
>
> "Dr. Christy having arrived [at Entebbe] should proceed along [the] west shore of Lake [Victoria] examining blood, and inquiring for Sleeping Sickness. Having got to the limit he should return to Mengo (Entebbe) and then start due west investigating in [the] same manner."
>
> "In this way the areas of Sleeping Sickness and *Filaria perstans* will be roughly determined in three directions. Then Dr. Christy should proceed down the Nile towards Egypt on the same quest, and if thought desirable should return home by that way."

## 5. Routes Followed.

(With Map.)

I left London on June 10, 1902, and, in company with Drs. Castellani and Low, arrived at Kisumu, the Victoria Nyanza terminus of the Uganda Railroad, on July 8. Here I parted with my two colleagues, who went on to Entebbe by steamboat, while I marched overland, through Kavirondo and Busoga, to the Nile at Jinja. From there I travelled the remainder of the way by canoe, reaching Entebbe on July 26.

Not having been able on this short journey to satisfactorily map out the eastern limit of Sleeping Sickness, it was necessary to make a second, and more extended journey, eastwards. For this purpose, I left Entebbe on August 6, and crossing the Nile at Jinja, travelled due north through Busoga, till Lake Kioga was reached, taking blood slides at intervals of from 15 to 20 miles. A little beyond Lake Kioga I found the northern limit of *Filaria perstans*, and touched upon the southern limit of *Filaria diurna*. I, therefore, at this point turned eastward to Mount Elgon. After passing along the western slopes of the mountain with some difficulty, owing to the hostility of the natives, I marched southwards through Kavirondo, taking blood slides at every village where possible, and reaching Mumia's on September 11. Thence I travelled on towards the Victoria Nyanza until I again entered the Sleeping Sickness area, where I spent some time in the worst affected districts, arriving at Kisumu again on September 20. Beyond Kisumu I travelled chiefly by canoe, mapping out the Sleeping Sickness area along the coast towards German East Africa. By arrangement with the authorities I was picked up by steamer at Lusinga

Island, and finally arrived at Entebbe on October 15. From there I made a visit of some days' duration to the badly affected islands of Buvuma and Bugaya.

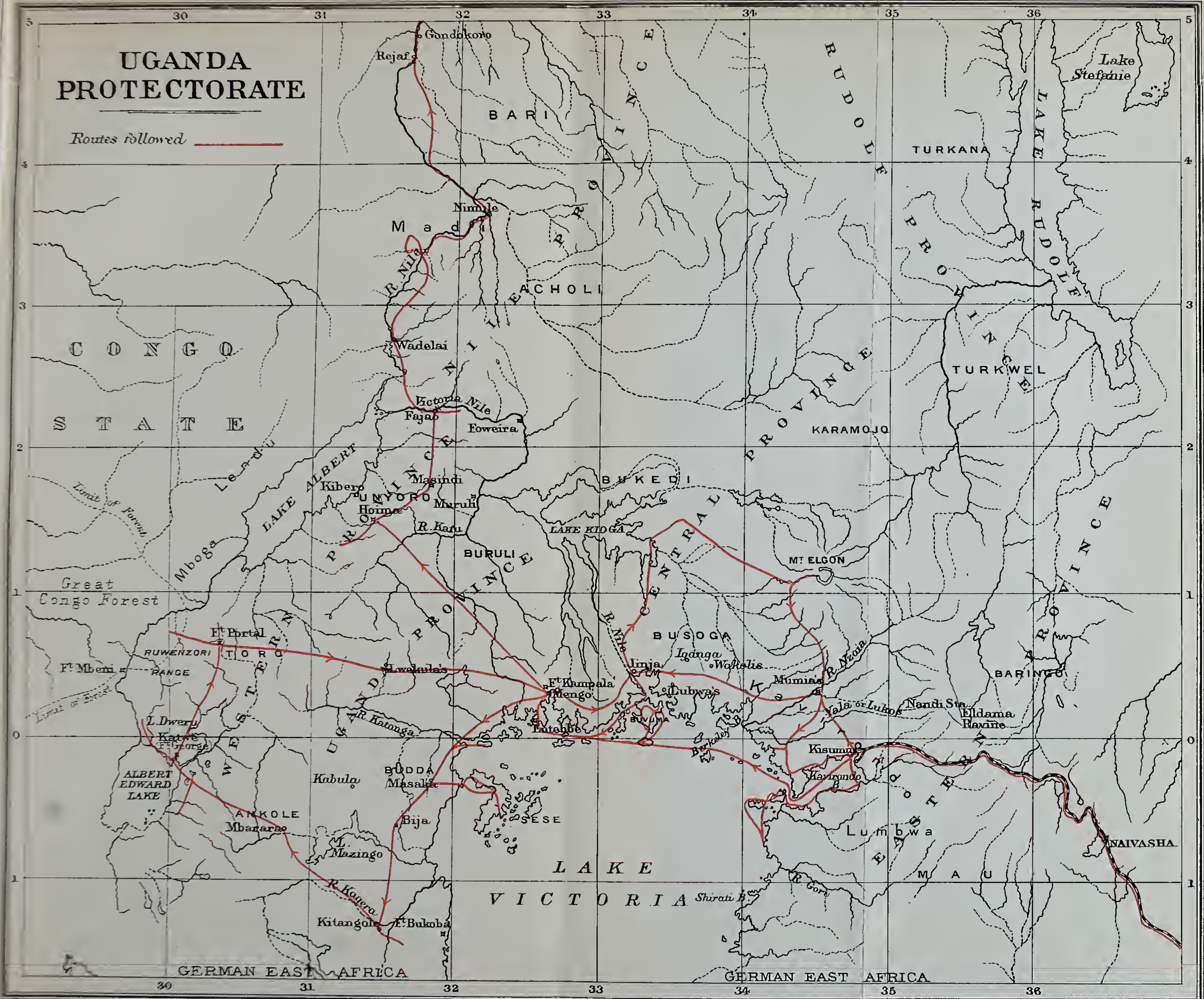
Leaving Entebbe, for my third and longest journey within the Protectorates, on October 30, I marched to Masaka in Buddu, from which place I paid a visit to the Sese Islands, and then travelled on southwards into German territory as far as the Kagera River, on the south bank of which, at Kifumbero (Kitangole), I had the pleasure of meeting Dr. Feldmann, the military medical officer stationed at Fort Bukoba, from whom I gathered much important information bearing upon Filariasis and Bubonic Plague in his district, as well as negative information with regard to Sleeping Sickness.

From Kifumbero I recrossed the Kagera and travelled north-west, through some difficult country, almost without tracks, to Mbarara, the capital of Ankole. Here I remained several days, and with the help of the collector of the district I was afforded an opportunity of explaining my mission to the King of Ankole and his chiefs at a full baraza convened for the purpose. Runners were immediately sent to all the surrounding districts, with instructions to the petty chiefs to send in at once so many men from each tribe, and I was thereby enabled to collect a large number of slides, and a store of information without traversing the whole of the province.

Leaving Mbarara I marched north-west till I reached the Albert Edward Nyanza, being considerably delayed on the way owing to the necessity of making treble marches for some days, the majority of my porters having run away. Skirting the eastern slopes of the Ruwenzori snow range I arrived at Fort Portal, the capital of Toro, on January 1. Here, through the kindness of the collector, I again had an opportunity of explaining my mission to the chiefs, and in the same manner as at Mbarara, obtained a large number of slides from the surrounding districts. I then travelled as fast as possible back to Entebbe, which I reached once more on January 31, 1903.

On arrival I heard that Dr. Low had already returned to England, and that the Royal Society wished me also to return. I had hoped after completing my maps of distribution to have been able to spend another month or so in further studying the etiology of Sleeping Sickness in Busoga and Kavirondo, and its more exact distribution along the margin of the lake.

After 5 days' rest, I left Entebbe *en route* for the Nile on February 6, 1903. On reaching Hoima, the capital of Unyoro, I learnt that owing to the steam launch on the Albert Nyanza being in use, it would be impossible for me to reach Gondokoro in time to catch the next monthly boat for Khartum. I therefore decided to march overland through Msinde,. Fajao, Wadelai, and Nimule, to Gondokoro, which place I reached on March 25. By so doing I was able to extend



Scale 1: 4,000,000 or 63·13 Miles = 1 Inch

0 10 20 40 60 80 100 Miles

my map of Filariasis, not only as far as the northern limits of the Uganda Protectorate, but also for a considerable way into the Sudan, and in addition to make sure that no Sleeping Sickness existed in that direction.

I went on board the river steamer at Gondokoro on March 27, arrived at Khartum on April 10, at Wady Halfa the 14th, Assouan the 16th, and at Cairo on April 18, 9½ months after landing at Mombasa. (See the route map.)

## 6. SOURCE OF THE OUTBREAK.

No medical officer was stationed in Busoga before July, 1901, and it is not unlikely that Sleeping Sickness may have prevailed in Busoga or in Buvuma Island for years before it was recognised by the Mengo Missionaries. All diseases are much alike to the native mind, and are regarded by him as visitations of evil spirits. The victim affects seclusion, and his superstitious friends make excuses for him. If there is much mortality a movement is made to another district, the people leaving their houses and contents, as well as the standing crops. Should this occur often the district becomes notorious, and an epidemic may be brought to light. Only when the people are hard pressed and their medicine-men have failed them, do they apply to the white man for relief. At all events from 1898—1900 there seems to have been a good deal of distress in Busoga and Buvuma Island, due to famine resulting from drought, and the consequent failure of the banana crop, and other food supplies. This may have to some extent masked a mortality really due to disease, or it is possible that famine or drought may have fanned into a severe epidemic what had previously been a mild and localised endemic, by affording the exact conditions required for the multiplication of the organisms causing the disease, or for the life and multiplication of some insect, or other agency, which conveys it. Spirillum fever in India, always endemic in certain areas, is apt to become seriously epidemic as the result of famine and drought.

Much importance seems to have been attached to the theory that the disease gradually spread, or was imported into Uganda from the upper waters of the Congo. It certainly has not spread across the intervening western provinces, either of Toro or Unyoro, but certain circumstances make it probable that it did come into Uganda from the westward. We know that between the years 1891 and 1895, a large number of Sudanese, with their women and slaves, were brought from the districts to the west of the Albert Nyanza, and settled in Busoga and Uganda under the following circumstances:—

After the relief of Emin Pasha by Stanley in 1888 at Wadelai, the ex-Sudanese soldiers of Emin's province established themselves at Kavali's to the west of the Albert Nyanza, under Selim Bey, living the life of freebooters. Upon the arrival of Captain (now General Sir Frederick) Lugard in Uganda, as the representative of the British East Africa Company in 1890, he soon realised, remarks Sir H. H. Johnston,* that if he was to play a dominant part in the settlement of Uganda, he must have a force of armed men at his disposal who were not Baganda. Accordingly, after the deposition of Kabarega, he proceeded (1891) to the Albert Nyanza, and from there recruited from Lendu and the surrounding districts 400 or 500 Sudanese, the remnants of Emin's army, bringing them, with a rabble of 7,000 wives, followers, and children, into South Toro, where he established them in a newly created kingdom, over which he had placed Kasagama, the present King, but owing to their lawless conduct, the greater part of them, with their locust-like wives and followers, were removed from Toro and placed under better control in Uganda and Busoga in 1892. Subsequently, during the years 1893—1895, many more of Emin's old warriors were recruited and brought, with their women and followers, to Busoga.

That some person or persons with Sleeping Sickness, or carrying the germs of the disease upon them, or that insects, arthropods, or other disease carriers, if there be such in this case, may have found their way into Busoga with these Sudanese, or their Lendu or Congo followers, is not improbable, but there is nothing to point to this theory being correct, except that the time of their immigration corresponds more or less with the probable commencement of the outbreak (1896). At Fort Portal, in Toro, however, I could gather no evidence to show that Sleeping Sickness existed, or had been known, anywhere in the country to the immediate west of the Albert Nyanza, although I questioned several natives and traders who knew that country well. Inquiries were also made for me amongst the oldest and most intelligent of the Sudanese, without gaining any record of the disease, and the officer who accompanied them was able to assure me that there had been no sickness amongst them, or their numerous followers, on the long tedious marches through Toro and Uganda. If the infection reached the Protectorate in this way it came with the followers, and not with the Sudanese themselves, for the latter are almost wholly immune to Sleeping Sickness.

On arrival in Busoga, the Sudanese warriors had no social intercourse with the native inhabitants, who were also looked down upon by the Congo and Lendu people, until after a few years a blood relationship gradually sprung up between them. Consequently, if the incubation period is a long one, as it is believed to be, it might be supposed that, if these people brought Sleeping Sickness with them, the disease would not be likely to appear among the inhabitants of Busoga until after the lapse of several years at least from the date of coming of these new arrivals from the west, and as a matter of fact, five years elapsed before the disease was reported by the missionaries at Mengo..

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* Johnston's 'Uganda Protectorate,' p. 236.

### 7. AREA OF DISTRIBUTION.

When the Commission arrived at Entebbe in July, 1902, Sleeping Sickness was thought to be widespread over Busoga and Uganda, but most prevalent in the former province, along the shores of the Victoria Nyanza.* My first journey through Busoga enabled me to satisfy myself that it was not only most prevalent, but was confined to the lake shores. On my second journey I traced it still further along the borders of the lake, from Busoga, through Kavirondo to Kisumu, and far beyond, down the eastern side.

The accompanying map (Revised Distribution of the Sickness) shows the distribution of the disease as I mapped it towards the close of 1902. The darkest coloured portions indicate what I have termed the infective area, or that in which the disease appeared to be contracted.

#### A. *Infective Area.*

This area consists of a narrow strip of coast line, at its widest not more than 10 miles, stretching along the shores of the Victoria Nyanza for a distance of over 250 miles. It extends from the mouth of the Katonga River, the northern boundary of Buddu, on the west, through Uganda, Busoga, and Kavirondo, to Kisumu on the east, the point at which the Uganda Railway reaches the lake. At Kisumu only two or three imported cases could be found, of which all but one had contracted the disease in Busoga or Uganda. Beyond Kisumu, along the eastern and southern shores of Kavirondo Bay, no cases were met with until reaching the Kasachonga and Homa districts. Passing Homa Inlet, I found that the Kasagunga coast district, opposite Uyoma promontory, and the large island of Lusinga, at the mouth of the bay, were badly infected. No cases were actually seen from Mfwangano Island. From Kasagunga the infective area extended southwards along the eastern shores of the lake, where I traced it to within a few miles of the Gori River. For information as to the occurrence of the disease south of the Gori River, I am indebted to Dr. Lott, the German Military Medical Officer stationed at Shirati, who wrote me on January 12th, 1903, saying that 4 months previous to that date, Sleeping Sickness had never been reported in his district, but that about that date it was said to have been brought by two natives coming from British territory. Altogether, four cases had occurred south of the boundary river Gori, in the Udemi district of the Sultan of Obo. One of these died two months previous to the date of his letter, the other three were isolated. No subsequent cases had come under notice.

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* Report 415, from Dr. Moffatt to Commissioner Sadler, Entebbe, June 17, 1902.

[Dr. Lott also stated that the natives reported that Sleeping Sickness was prevalent in our districts of Kabotschi and Unjama, north of the Gori river.* A similar report was made to me by the Kasachonga people, and the Kisengere people said that there were a few cases a little way inland. Also at Nyacach, at the head of Kavirondo Bay, the chief informed me that he thought the disease was prevalent in the Lumbwa country, and he pointed quite correctly to the south-east, saying it was two days' journey. As the infective area shows nowhere else any tendency to spread beyond a very short distance inland from the shores of the Victoria Nyanza, the above reports of the occurrence of the disease towards the Mau country are of some interest, and I regret that during my visit, I was not able to investigate the matter further. I had not then received Dr. Lott's letter, and I was obliged to be at Lusinga Island upon a certain date in order to meet the steamer on her way to Entebbe. The Lumbwa country was, too, at that time practically unexplored, and to have made a journey through it would have necessitated an expedition on a much larger scale than the Protectorate authorities would have been prepared to sanction.]

The infective area also includes all the islands in the British portion of the Victoria Nyanza, and, judging by investigations I carried out in the Sese and Buvuma groups, it is in the larger islands, probably confined to their margins, in the same way as it is confined to the lake shores on the mainland.

I was not able to visit many of the smaller islands, but I was informed by missionaries and others that all of them were more or less affected, Kome Island the most seriously. The White Fathers of Vitala Maria assured me that in Kome Island even cattle and dogs had died of the disease.

With regard to the western limit of the infective area only a very few cases were to be found or heard of west of Entebbe and Kampala, and none south of the Katonga river, with the exception of three cases at Masaka, which there was good reason to believe had been imported; two cases at Bujaju, the nearest mainland to the Sese Islands, both of which had come from Sese; and one rather doubtful case at Burangoma, a small village not far from Charibika on the hills overlooking the south end of Lake Mazingo, in Ankole. I could gain no information that this case, a boy, had ever left his native village. His father stated that there were two similar cases, but they were not forthcoming, and I was hampered in my search by the want of an interpreter.

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* On Lieut.-Col. Macdonald's latest map, issued by the War Office, which I used during my journeys, the hill district of Kaboshi (doubtless the same as Kabotschi) is marked as being 15 or 20 miles from the lake shore.

Although I travelled, during my 9 months' stay in the country, over the greater portion of the better-known districts of the Uganda Protectorate, as well as the western parts of the East African Protectorate, and into German East Africa, and the Congo Free State, making inquiries of the chiefs and people, coupled with personal investigations, I found no other endemic areas of Sleeping Sickness beyond those on the shores of the lake and in the islands. It does not occur on the Upper Nile or its tributaries, and there seems no reason to believe that it has spread to the Nandi Plateau as has been stated.

### B. *Non-infective Area.*

The lighter coloured portions of the Distribution Map, namely, those parts beyond the infective area on the mainland and the interior of the larger islands, indicate the area in which the disease is prevalent, but in which it does not seem to be infective. I have called this the non-infective area. The cases occurring in it are usually isolated ones, scattered here and there, and whole families are never infected. Moreover, in the great majority of these cases a history of the patient having been at or near the lake some months previously can be obtained. I found or heard of a few cases even beyond this area, as far as Tabingwas in Busoga, but all of them had been to the lake. The natives themselves in some districts recognise that the disease is contracted only in districts from which the lake can be seen, and infected individuals frequently volunteered the information that they had been to the lake.

## 8. INHABITANTS OF THE INFECTIVE AREA.

The area of distribution of Sleeping Sickness in Equatorial Africa is about equally divided between two of the most widely differing types of negroes as could be found in Tropical Africa. To the west of the Usoga-Kavirondo boundary are the provinces of Busoga and Uganda, inhabited by a clothed and fairly civilised Bantu race. To the east of the Usoga-Kavirondo boundary are the Kavirondo, typical Nilotic negroes, unclothed and uncivilised, with language, customs, food, and appearance entirely different from their neighbours to the west of them.

The inhabitants of the islands are in most cases of a type similar to those of the mainland opposite to each.

## 9. PHYSICAL FEATURES, AND CLIMATE OF THE INFECTIVE AREA.

The Victoria Nyanza is 3,775 feet above sea level. Along most of the northern coast, from the Katonga River on the west, to Berkeley Bay at the Busoga-Kavirondo boundary on the east, the catchment area of the lake approaches to within 5 or 10 miles of its shores, for in this section practically no running streams enter it and only the Nile flows out of it. This portion of the Victoria Nyanza is bordered by hills, for the most part thickly wooded, and rising 500 or 600 feet above the lake level.

The lake shore itself, is in most places low lying and forested, and in inhabited parts it is planted with bananas. Only in the more secluded inlets is there any considerable collection of papyrus, and marsh. Kavirondo is mostly characterised by rolling upland downs, but the coast, here also, for a few miles inland is low lying, and where the Nzoia and Yala rivers enter the lake there are considerable growths of papyrus.

At the head of Kavirondo Bay there is a large area of swamp, at the edge of which are several big villages, inhabited by Kavirondo fishermen, who at the time of my visit had not been attacked by Sleeping Sickness. The south shores of the bay, and the eastern shores of the Victoria Nyanza, are bounded by high grass-covered hills sloping gradually to the lake, with occasional patches of open forest.

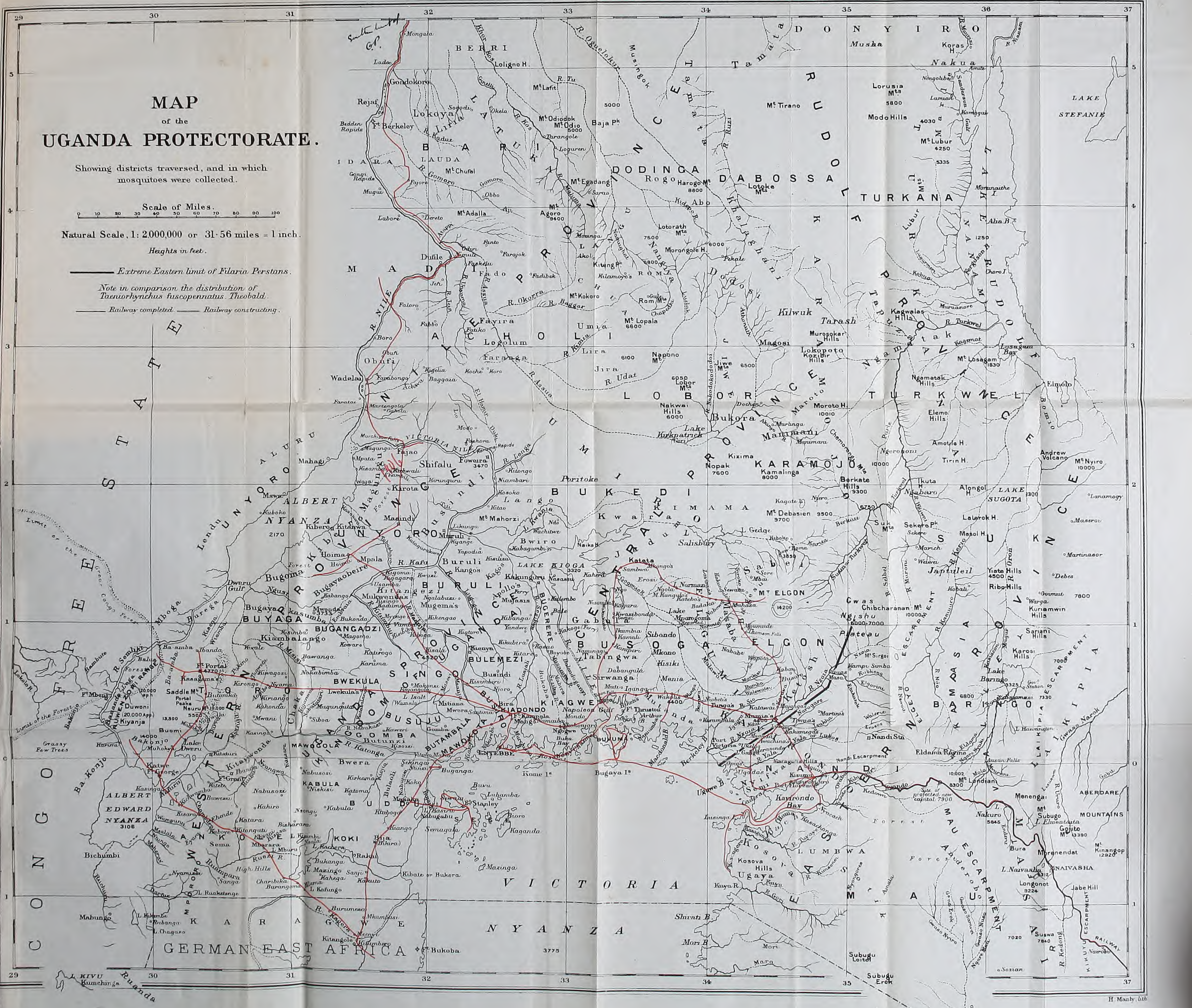
The majority of the islands are high and rocky, and are covered with vegetation; some have patches of magnificent jungle forest in the valleys, and round the margins.

The sun at Entebbe rarely causes any discomfort. The average minimum temperature is about 52° F., and the maximum 81·8° F., and, although it is situated less than 10 miles north of the equator, its climate is much like that of an English summer.

## 10. CHARACTERISTICS OF INFECTIVE LOCALITIES.

In travelling through the infective area it was frequently noticed that Sleeping Sickness did not affect the inhabitants of all places alike.

The disease was most prevalent amongst the inhabitants of low-lying shambas (banana and potato plantations) in places along the shores of the Victoria Nyanza, or in wooded districts not far from the water. But even in these districts natives who lived in villages, particularly if they were situated on high ground, were much less affected than those living in the shambas; and those living in large compact villages, encircled by the usual high cane structures, were less seriously affected than the occupants of scattered villages in which the huts were surrounded by bananas or forest growth. People inhabiting populous



# MAP
of the
# UGANDA PROTECTORATE.

Showing districts traversed, and in which mosquitoes were collected.

Scale of Miles.

Natural Scale, 1: 2,000,000 or 31·56 miles = 1 inch.

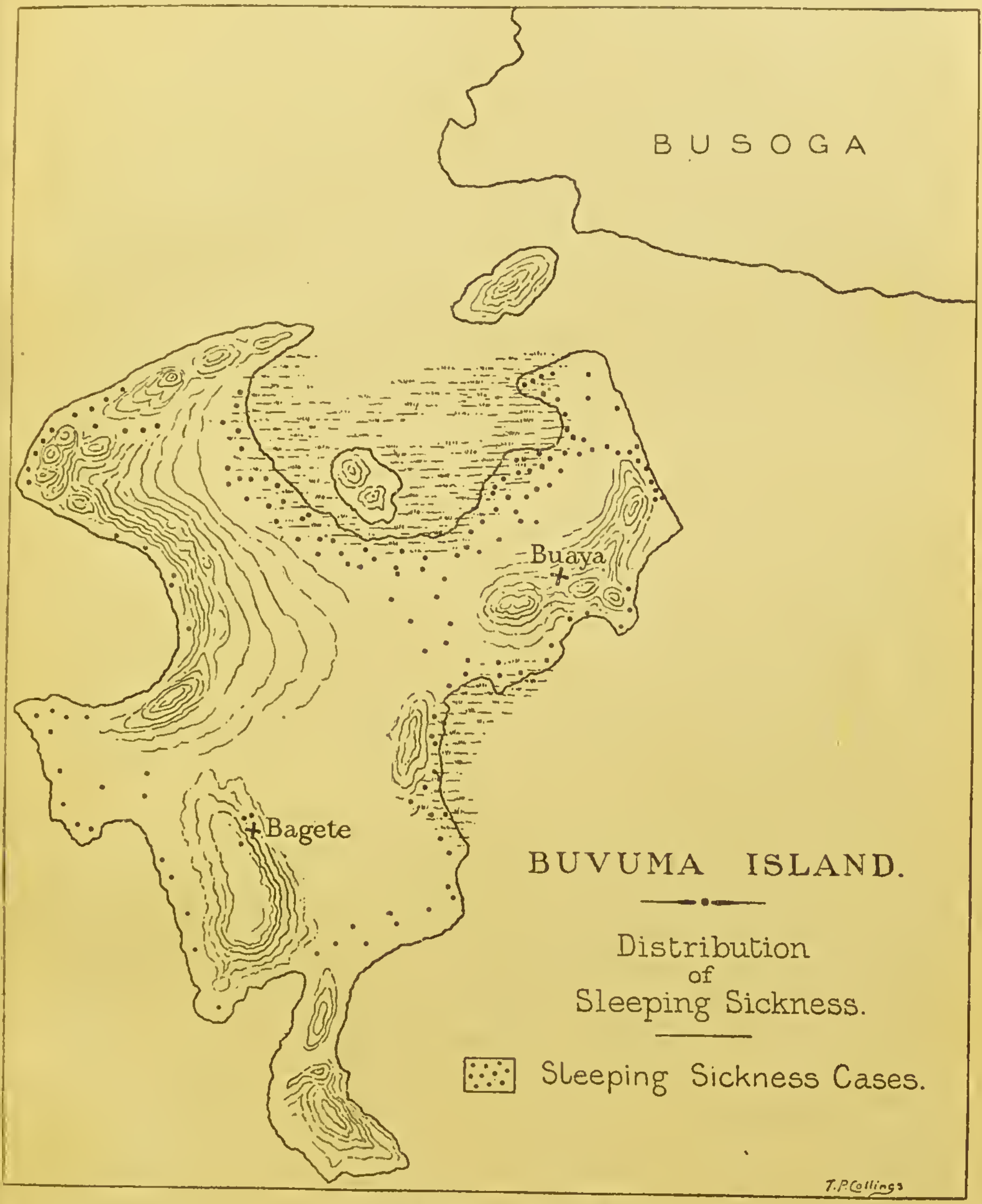
*Heights in feet.*

——— *Extreme Eastern limit of Filaria Perstans.*

*Note in comparison the distribution of Taeniorhynchus fuscopennatus. Theobald.*

——— *Railway completed.* ——— *Railway constructing.*

centres, such for instance, as Kampala and Entebbe, were practically exempt, while those living on open grass plains were quite free from the disease.



(Compare Plates 1 and 2.)

In order to study more closely cases of Sleeping Sickness within a circumscribed area, I made a visit of several days' duration to the badly infected island of Buvuma. The accompanying map illustrates one result of the investigation. I was conducted from shamba to shamba all over the island, and found that a large proportion of the huts were empty owing to the death of the occupants, while many of the inhabited ones contained a case or cases of Sleeping Sickness. Practically all the cases were confined to the lower levels of the island, near the water's edge, and the dwellings were surrounded by bananas or forest growth. The village of Buaya, situated on high open ground, did not contain a single case, and only two deaths had occurred amongst the people in the chief's village of Bagete, situated fairly high, but adjacent to forest growth. These characteristics of infected localities in Buvuma held good for all the infective area on the mainland, as far as I was able to observe; for example, at the Roman Catholic Mission station of Nkokonjaro (Kiagwe, Uganda) the Fathers stated that many deaths from the Sickness had occurred in the shambas down near the water, but not amongst those living round the Mission, which was situated on high ground, a mile or two from the lake.

In marked contrast to Busoga and Uganda, the prevailing landscape in Kavirondo consists of rolling downs covered with short grass. The infective localities are (as elsewhere), the districts bordering upon the Victoria Nyanza, and the estuaries of the rivers, where scattered trees and scrub appear, with fantastic candelabra-shaped euphorbias. In Kavirondo, however, the exemption of villages from the disease was less noticeable than it was to the westward, for the reason that nearly every family had a separate village surrounded by a high cane wall, or an enormous floral hedge, and single huts were rare. Here, again, the villages which were situated on high ground and in the open, although within the infective area, were almost exempt, the majority of cases coming from the low-lying scrub country adjacent to the lake and the river estuaries.

It is remarkable that the large number of Sudanese who were brought from Lendu and the Congo (see "Source of the Outbreak") remain to the present time practically free from Sleeping Sickness, although in many instances they have been quartered for years in the infective area. The reason for this lies probably in the circumstance that they live in large, well laid out villages. These "Nube lines," although filthily insanitary on the outskirts, are exceedingly clean and neat within. The huts are in rows, and no vegetation is allowed to grow up near them. The streets are 10 or 20 feet wide, and are swept daily. Each man has several women belonging to him, whose duty it is to keep the floors and doorway carefully and regularly cow-dunged once a week or oftener. Doubtless the partial exemption of these Sudanese from *F. perstans* may be accounted for in the same way. Many of the slaves and people who followed them into Busoga reverted to their original condition as workers, and they or their descendants are to be found living amongst the shambas in the neighbourhood of the lines. These have suffered as much from Sleeping Sickness as the original inhabitants of Busoga.

## 11. PROGRESS OF THE EPIDEMIC—DIRECTION OF SPREAD.

At Jinja Dr. Hodges informed me (July, 1902), that he believed that the original starting point of the epidemic was Wakolis in Busoga, and he stated that native report said that it had commenced there six years ago (1896). When I passed through Wakolis, which is situated some 15 miles from the Victoria Nyanza, and well outside the present infective area, the old chief insisted that there were then no cases of Sleeping Sickness, and I found none, but there had been cases some time previously. What spot was first infected it is now impossible to say, but in all probability the epidemic did start somewhere in Busoga, or in the neighbouring island of Buvuma.

From Busoga the epidemic has travelled east and west, both along the mainland and the chain of islands. Westward, on the mainland, it seems to have spread with some amount of virulence as far as Kampala and Entebbe, but beyond these places little was to be heard of the disease, and only a few indigenous cases were to be found, as far as the Katonga river, where the infective area apparently ceased. It seems, however, to have spread with much more virulence along the chain of islands westward until it reached the Sese group. The White Fathers in Sese stated that they believed the infection had been brought from the islands to the north, with which there was constant communication. The infection certainly could not have reached them from the adjacent Buddu coast, where, with the exception of imported cases, the disease was not to be found. The White Fathers also stated that the first cases in Sese occurred 11 months prior to my visit (November, 1902).

I was not able to visit many of the islands besides Sese and Buvuma, but several of my informants were of opinion that those off the Kiagwe coast, especially Kome, were infected some time before the mainland, which I think is probable, because a good deal of traffic exists between the islands, but less between them and the mainland.

Eastward, from Busoga, the epidemic spread along the mainland and the islands into Kavirondo, with much greater virulence than it had shown in travelling westward, and when I left Uganda it was extending its area down the eastern side of the lake. The first cases in Southern Kavirondo occurred 11 months prior to my second visit, and the natives believed that it had been introduced by Busoga canoe men.

On reaching the infected regions south of Kavirondo Bay (September 26, 1902) I learnt that in Kasagunga the first cases had occurred about 9 months previously. That the epidemic had crossed the mouth of the Bay at its narrowest part seemed to be proved by the statements of the natives that the first cases in the districts (Lusinga Island and Homa) on each side of Kasagunga, which

occur in villages are of the poorest class, and work in the fields all day.

*Chiefs*, and the majority of the better class natives, live in the villages, and consequently are less affected by the disease. The Sub Commissioner for Busoga could only give me the two following instances of chiefs having died of Sleeping Sickness. Mbanda, a regular visitor to Jinja Fort on Sundays, was one day noticed to be asleep during the holding of a court, and when taxed with inattention his people admitted that their chief had "Mgota." He died of the disease 6 months afterwards. The petty chief Lukalanga (an incorrigible drunkard) succumbed after only 4 months' illness.

### 13. INFLUENCE OF SEASONS, FOOD, AND DRINKING WATER.

I could not trace any connection between Sleeping Sickness and the wet and dry *seasons*, nor could I gather any information from the natives which pointed to the disease being more prevalent at any particular time of the year, and the fact that the duration of the attack extends over several months would make this unlikely. The wretched victims, whose sensibility to cold is much increased, are perhaps more in evidence during the hot dry weather, for they are then induced to crawl out of their huts and lie in the sun.

There is no article of *food* used in the infective area which is peculiar to it. The Baganda live almost wholly upon bananas, which are picked green, peeled, and steamed in an earthenware pot over a slow fire. The staple article of diet of the Kavirondo is grain, chiefly "dura" (millet), to all appearance the same plant as the Indian "duari." It is ground into flour and made into a sort of porridge, or into dampers and cakes. The Kavirondo will also eat almost any kind of meat or dried fish, and in the cattle-grazing districts quantities of milk and butter are consumed.

Dried fish is the only article of diet used throughout the infective area. The smaller fish, the young of a species of perch,* are threaded on sticks and dried in the sun; the larger fish (mostly of the same species) are opened, cleaned, and smoked over a wood fire. I have seen these cured fish all over the country far outside the Sleeping Sickness area. Fish have been suspected by the medical officers of the Protectorate, as well as by the natives, as the cause of the disease, because of the close association of the latter with the shores of the Victoria Nyanza. If fish-eating were the cause, the province of Buddu would not be exempt, and Europeans would before now have become infected.

That the Victoria Nyanza has some very direct influence upon the disease is clear from a study of its distribution, but that it is caused by drinking the *water* of the lake is highly improbable, for many of the inhabitants of the infective area get their supply elsewhere, from streams, ponds and springs, while at Entebbe, which is practically surrounded by the lake, both Europeans and natives use the lake water for all purposes, yet the disease is not to be found in the town itself.

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* Identified by Mr. Boulenger, of the British Museum, to whom I submitted a specimen, as *Talapia galilæa*.

## 14. Propagation Characteristics.

A very little observation shows that Sleeping Sickness is not contagious, as we usually accept the term, and that it is only infectious in a limited sense. A person suffering from the disease, who travels to a district beyond the known infective area, does not convey it to persons in that district unless the conditions, whatever they may be, are favourable to its propagation, and that district must apparently be one bordering upon the Victoria Nyanza. If, however, an infected person travels from one part of the infective area to another, or into regions beyond, which, although previously uninfected, contain the conditions favourable to its propagation, then he becomes a source of danger to all with whom he comes in contact, much in the same way probably as a person suffering from malaria. As is known, if the malarial subject reaches a camp or district in which a suitable species of mosquito occurs, then, at a certain stage in his attack, he becomes a danger to those around him, and still more so to those who come after him. If, however, he camps in districts not inhabited by a suitable mosquito, he may have recurrent attacks with impunity to other people. Without a more competent interpreter than I had, it was impossible for me to ferret out sufficient detail to make a reliable computation of the dates at which the various members of a household or of a district became infected, and of their relation to each other, but the sum of my investigations has led me to believe that the method of propagation is somewhat similar to that of malaria.

Throughout the non-infective area one hears of deaths of persons who had contracted the disease within the infective area, and cases are met with which have developed the disease months after leaving that area. They are almost invariably single cases, which do not seem to have infected other persons in the household or the neighbourhood, and they cause no alarm.

The following is the best authenticated example, amongst several related to me, of the infectiousness of Sleeping Sickness within the infective area, and of its non-infectiousness outside that area:—

Overlooking the Ripon Falls is Jinja Fort, built by the Sub-ommissioner for Busoga, amongst whose people were three young girls A., B., and C., who had been there, he told me, since 1898.

They all three occupied the same room. B.'s parents lived near the Mpologoma River, 60 miles or more from the Victoria Nyanza. C.'s home was a shamba near the lake. In July, 1900, B. and C. went to C.'s home, the shamba near the lake, for a month's holiday. About March, 1901, 8 months afterwards, C. was noticed to be peculiar in her manner. She laughed and cried without cause, talked queerly, and seemed very weak. She was, therefore, sent home, and subsequently caused her people a great deal of trouble through her antics and annoying conduct. Eventually she became too weak to stand, and died 3 months after reaching her home. All her family have since died of Sleeping Sickness.

B. was sent to her home, 60 miles from C.'s home at the lake, suffering with the disease, in January, 1902, 18 months after visiting the lake. She was noticed to be very quiet, said very little, and was drowsy. She died 4 months after reaching her home. For a month previous to her death, she was unable to speak, and if spoken to would open her eyes and perhaps make signs. She could neither stand nor walk, but her faculties were clear till death. Her people were all healthy, and no other cases had occurred in the district (September, 1902).

A., who had lived in such close contact with these two girls, sleeping with them in the same room for so many months, both before and after B. and C. were known to be infected, was perfectly healthy at the date the story was related to me. No other cases of Sleeping Sickness occurred, either before or since, in the fort.

The Sese Islands, about 5 miles distant from the Buddu coast, have been badly infected since the end of 1901; and yet, although there existed, in spite of precautionary measures, a considerable amount of traffic across the intervening portion of the lake, the epidemic had not spread to Buddu, the necessary favourable conditions apparently being absent there. On the other hand, the epidemic, comparatively early in its history, as soon as it reached the Uyoma promontory, in its rapid extension eastwards, crossed the mouth of Kavirondo Bay, a distance of 3½ miles, into the Kasagunga district, although the traffic from shore to shore at this point was, according to native testimony, very infrequent. In this case the necessary conditions for the propagation of the disease must have been present on the southern shores of the bay, the disease itself being probably brought over by some infected Basoga or Kavirondo native, for the people in Kasagunga and Lusinga Island would not admit that they themselves ever went across.

The infective area does not spread along caravan tracks or roadways, and the occurrence of four imported cases at Mumia's, in Kavirondo, and two or three cases along the main road from Busoga to that station, was the only instance I met with in which a road contributed to the spread of the disease, but in this case it only aided the transport of infected individuals, and not the extension of the infective area. Nor does it show any tendency to spread along the course of rivers beyond their estuaries. The disease was prevalent at Ripon Falls—the "birth of the Nile"—but I could not gather that it had extended for more than a mile or so northwards along that river. It is not found, as far as I know, on the shores of any of the other great lakes.

During the early stages of my investigation I formed the belief that the infective agent, whatever it might be, existed in the huts of the natives, but I subsequently saw reason to modify my views upon this subject. If it were so, the houses of the White Fathers, which are usually large, sun-dried brick structures, could not escape infection, for the missionaries live in very close relations with the natives, their churches and mission houses being crowded daily with men, women, and children, some of whom are probably infected; moreover, they visit cases which occur in the shambas, sit with them, and attend to their wants, yet there is no record in Uganda of a missionary, or any European, having contracted the disease. In the Semi district, of Kavirondo, where the disease was much more virulent than to the westward, 30 or 40 per cent. of the inhabitants being infected, and where missionaries are unknown, I camped several times, as malaria seemed rare, in the midst of huts containing Sleeping Sickness cases, and spent days, both in Semi and Buvuma, turning out their contents, sketching, photographing, and examining implements and utensils. There seems, therefore, strong presumptive evidence that the infective agent is at any rate not a blood-sucking insect or arthropod inhabiting the huts.

All the evidence I have been able to gain points to the infective agent being something which frequents the low-lying shambas, and the forested or scrub-covered country along the shores and inlets of the Victoria Nyanza and its islands, but the question arises, should this view be correct, why it is that missionaries, and other Europeans, who have occasion to pass through or spend their time in shambas within the infective area do not become infected? Possibly, in their case, clothing acts as a protective. With a view to seeing whether mosquitoes were implicated, I made a large collection of them in all parts of the country traversed. Since their identification, I have made a study of the distribution of each species, and it is clear that there is no species whose distribution in any way corresponds with that of Sleeping Sickness.*

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* See Report on the Collection by F. V. Theobald, Esq., M.A., B.Sc., p. 33, *infra*.

## 15. IMMUNITY, AND INFLUENCE OF RACE.

The fact that the Nubian police, the remnants of Emin Pasha's Sudanese, are practically immune to Sleeping Sickness, as already mentioned, would seem to indicate that race may have some influence upon the disease. This, however, is probably not the case, for the infective area is divided between two widely different races of negroes (see section "Inhabitants of the Infective Area"), those on the east of the Busoga-Kavirondo boundary being closely related to the Nilotic races from which the Nubian police were recruited, yet their kinship has not saved them.

The people in Buvuma Island asserted, and it was well known, that the Baganda living in the island were not seriously affected by the disease, whilst the Bavuma themselves were decimated by it. But the explanation is probably to be found in the fact that the Baganda, the conquerors of the Bavuma, belong to the better classes and live in the villages, while the original inhabitants live and do most of the work in the shambas.

It is possible that race influence may have some bearing upon the length of the incubation period and the character of the symptoms, but upon this subject I can give no definite information.

## 16. SEVERITY AND DIMINUTION OF THE EPIDEMIC.

At what I may call the head of the epidemic, or that district in which the disease was most virulent, namely, in Kavirondo, and to the eastward, not only were cases more numerous, but their duration was much shorter, and the symptoms far more acute, than in Busoga and Uganda. In Kavirondo, hysterical excitement, violent mania, and harmless dementia, were common, and the duration averaged between 4½ and 5 months. In Busoga, and Uganda, the disease had considerably diminished when I passed through, both in number of cases and severity, and the duration was from 6 to 8 months, even 13 and 14 months was not uncommon. Here also the majority were not typical cases, somnolency being frequently almost absent until the late stages developed.

## 17. *Filaria perstans.*—ITS CONNECTION WITH SLEEPING SICKNESS—ITS DISTRIBUTION, ETC.

On reaching Uganda it was my first aim, as instructed, to map out the area of distribution of Sleeping Sickness, and afterwards that of *F. perstans*, in order to see if the two areas in any way corresponded, as they were supposed to do.

Travelling eastwards from Entebbe it was found that all through

Uganda and Busoga, *F. perstans* was common. From 60 to 80 per cent. of both men and women harboured the parasites; but once past the Busoga-Kavirondo boundary the percentage dropped abruptly to 14 per cent. and then to 5 per cent., a fact first noted by Dr. Hodges, who, however, was not able to travel far enough beyond his district into Kavirondo to reach the limit. Still further east, beyond the Nzoia river I found that *F. perstans* was entirely absent, even in districts where Sleeping Sickness was affecting nearly half the population. I took several hundred blood slides in Southern Kavirondo east of the Nzoia river without finding a single embryo. My discovery (September, 1902) of the total absence of *F. perstans* in Southern Kavirondo was afterwards confirmed by Dr. Wiggins, then recently appointed medical officer at Kisumu, who, after travelling with me along the south of Kavirondo Bay, to the Kasagunga district and Lusinga Island, on his way back to Kisumu, made his first journey through the parts infected with Sleeping Sickness on the north of the bay, and collected his first blood slides. He wrote me (October 12) that he had taken many slides, but found no *F. perstans*. The total absence of *F. perstans* prevailed, I found, far beyond Kavirondo Bay, and down to the eastern shores of the Victoria Nyanza; it probably does not occur anywhere in the East African Protectorate, westward of the Nzoia river.

This important discovery, that Sleeping Sickness extended along the lake shores for more than 100 miles beyond the extreme easterly limit of *F. perstans*, enabled me to send to the Society, on October 31, 1902, the first proof that the two diseases are not connected with each other.

The area of distribution of *F. perstans* extends over the whole of Busoga, Uganda, Buddu, part of Ankole, Toro and Unyoro, as far as the Albert Nyanza, 150 miles north-west of the Sleeping Sickness area. I also traced it into the Congo Free State, and found a few cases on the Nile at Wadelai, and again at Gondokoro. If *F. perstans* were the cause of Sleeping Sickness the disease would have spread far and wide from the shores of the Victoria Nyanza.

Again, in Buvuma and other places I took a large number of blood slides, both from Sleeping Sickness cases and from persons apparently free from the disease, and found that the percentage of *F. perstans* in each case was practically equal; I have yet to complete the compilation of some statistics upon this matter.

It is interesting to note that I found three spots in the Protectorate, far outside the Sleeping Sickness area, where the blood of the natives was so full of embryo filariæ that 100 or more of them could be counted in most of the slides, and yet there were no signs of Sleeping Sickness amongst the inhabitants. On the other hand I found no place in the Sleeping Sickness area where anything like so large a number could be found in any one slide.

During my tour through the lake regions, and for a considerable distance down the Nile into the Sudan, I was able to collect blood slides at the majority of my camps, as well as from natives sent to me from outlying districts by arrangement with collectors and chiefs. The total number of persons, whose blood was examined microscopically for *F. perstans*, amounted to over 5,800, from 155 different places, a success far beyond my original expectations, for I had to travel through several districts only visited previously by one or two Europeans, and some also in which the natives were hostile. With the material gathered I have been able to completely map out the areas of distribution, in British territory, not only of *F. perstans*, but of other species of Filaria, and also to collect a large amount of information bearing upon the general subject of Filariasis.

## 18. CLINICAL OBSERVATIONS.

I do not enter minutely into the clinical features of Sleeping Sickness, for this part of the work of the Commission was undertaken by my two colleagues, who remained in Entebbe, and had many cases under their care in a special hospital. As, however, during my several journeys, I had opportunities of studying a very large number of cases in their own homes, amidst native surroundings, the following notes may be of some interest:—

With regard to the *incubation period* I have shown (see "Propagation Characteristics," histories of A., B., and C), that it may be as short as 8 months, or as long as 18 months. Little is known as yet of the *premonitory symptoms*, but I may state that towards the termination of the incubation period a gradually increasing lassitude and weakness of the limbs occurs, and afterwards frontal headache.

### A. *First Stage—First Month of the Attack.*

As the attack developes the headache increases, the patient becomes morose and slow in his movements, is disinclined to work, and wears a pained, frowning expression, which can be at once recognised as absolutely characteristic of the sickness at this early stage. Later, a certain vacancy is added to his expression, the lower lip becomes puffy and drooping, the general weakness increases, he continually wants to sit down, and frequently dozes, he complains of pain at the back of his neck, in the ankles and elsewhere, his tongue is tremulous, and his hand grip is weak and unsteady.

### B. *Second Stage—Second Month of the Attack.*

In this stage the drowsiness and weakness go on increasing, the face becomes puffy, the lower lip more swollen and drooping, the expression vacant, almost idiotic, the skin dry and scaly, speech thick and husky, marked tremors in the muscles develop, frequently more noticeable on the left side, the movements of the hands are jerky, an apparent heaviness and drooping of the upper eyelids is conspicuous, and saliva dribbles from the corners of the mouth or over the puffy lower lip. As this stage progresses the whole body becomes puffy and bloated, as well as the face, and somnolency is a troublesome feature. (*Cf.* Plate 3.)

### C. *Third Stage—Third and Fourth Months of the Attack.*

During the third stage the symptoms are all intensified, the weakness becomes so great that the patient is obliged to walk with the help of a stick, and often, instead of sitting down, falls in a heap without attempting to save himself. The somnolence may now be almost continuous, the skin, from want of attention and weakened vitality, has become covered with craw-craw and filthy, and the tremors in many cases are choreic. As time goes on the puffy, bloated appearance of the body subsides, emaciation sets in, and he lies on his side with his limbs flexed, oblivious of everything around him, utterly unable to help himself, and only returning to partial consciousness if roughly shaken.

### D. *Final Stage—Fifth or Sixth Month of the Attack.*

During the final stage (Plate 4) the wretched being becomes emaciated and filthy in the extreme, extensive ulcers develop on the hips, ankles, elbows, and other parts, urine and fæces are passed under him, and he lies in a deeply lethargic condition until death supervenes.

This will give some idea of the progressive stages of the disease as I saw it in Kavirondo, where the epidemic was most severe, the duration of the cases comparatively short, and the symptoms well marked. To the westward, in Busoga and Uganda, where the epidemic had to some extent diminished, each stage of the disease was lengthened out, all the symptoms were less marked, and some of them were absent.

In Kavirondo, violent excitement, and other head symptoms, at the very beginning of the attack, seemed more frequent than elsewhere. At one of my camps in that country, a powerfully-built man, with the characteristic facies of the earliest stage of the disease, whom I had noticed hanging round the outskirts of a crowd of natives, amongst whom I was taking slides, suddenly made a leap into the crowd and struck out right and left with a native hoe, creating a momentary panic, during which my table and slides were upset. He was soon overpowered by his friends, however, but not before he had cut open the heads of two of them, and was carried away struggling and shouting to a small village close by, where I heard him raving till the early hours of the morning. On questioning the people, they stated that he had been quite well till three days previously, when he had had a similar attack. During the struggle he was in a state of tense priapism.

Cases of crying and laughing, and weak hysterical excitement, were, perhaps, more common in the second stage, and were frequently seen elsewhere than in Kavirondo. Nystagmus I never observed. The muscular tremors increase gradually from an early stage, occasionally distinct rhythmic waves of contraction can be seen running from one end of a muscle to the other. A high pulse rate is a nearly constant feature throughout the attack, and, I believe, an early symptom. Puffiness of the face, and particularly of the lower lip, is also a constant feature; in many cases the tongue is swollen and protruded (see Plate 3), and the eyeballs prominent, as in exophthalmic conditions.

The distension of the abdomen, and the puffy bloated appearance of the whole body in the second stage, in the majority of cases, gives place, during the third stage, to wasting and emaciation, particularly in Kavirondo. In Buvuma and the Sese Islands, however, where this bloated appearance was far more common than elsewhere, some of the cases showed no signs of emaciation, not even in the last stage, and somnolency in them was not a marked feature. In these cases also the appetite, which always remains good till the third stage, when weakness and somnolency gain the upper hand, might be described as ravenous, and remains so till death occurs. In Buvuma and Sese Islands I saw cases in the last stage so weak that they could hardly lift a hand, and yet were so bloated as to appear only in the second stage of the disease. They were drowsy but not somnolent, and their friends complained that they could not get enough food to satisfy their inordinate hunger.

Many cases, particularly those of the bloated type, when sitting on the ground adopted a characteristic attitude. The legs were separated and extended in front of them, the arms hung by the side, the head was bent forward, and the ropy saliva dribbled down the distended belly, or streamed on to the ground between the legs.

Symptoms of a peculiar nature, bordering upon a cataleptic condition, were frequently to be seen. For example, a patient who yawned and stretched his arms would sometimes forget to withdraw one of them, and it might remain in its extended position for 10 minutes or more; or he would be in the act of feeding himself, and, instead of bringing his fingers to his mouth, would doze off and let the hand remain half-way up to his face for some minutes, or until his next interval of wakefulness. In Buvuma an old and very emaciated woman, who died while lying in front of my tent, held up her arm, in a position possibly intended to shade her face from the sun, without moving it for fully an hour before she died.

A particularly intelligent native chief commented on the disease as follows: "Mgota comes very slowly, it catches one man and he dies, then catches another man who dies, then another. It makes one man very fat, another very thin, and some to eat very much."

### *Glandular Enlargements.*

The early records relating to Sleeping Sickness mention enlargement of the cervical glands as having a connection with the disease, and there is no doubt that there is some intimate association, for I have discovered that the area of distribution of enlarged cervical glands in Equatorial East Africa agrees with that of Sleeping Sickness. During my four journeys in the Protectorates I examined the neck of every man, woman, and child, from whom I took a blood slide, for enlarged glands, and from the extensive tables I have in hand, compiled from this material, together with the results of the examination of many hundreds of natives from whom I could not obtain slides, or had sufficient, I am able to show that the two areas practically correspond. The region of enlarged cervical glands stretched inland a little further than that of the non-infective area of Sleeping Sickness, and from that point the proportion of enlarged glands in the general population increased as one neared the Victoria Nyanza, the highest proportion, together with the greater enlargement, being on the islands. Beyond the agreement between the two areas, there seemed no further connection between Sleeping Sickness and enlargement of glands, for although the majority of cases of the former had firm, and freely movable, glandular enlargements in the neck, and some of them all the superficial glands in the body enlarged, yet there were many cases with no enlarged glands whatever. Again, in several places within the infective area where I was able to line up in front of me several hundred natives, I first picked out all those whose pulse rate was above normal, and then examined them for enlarged cervical glands. Here, also, there seemed no agreement, for some of those selected were without any enlargement, whilst many of those who showed no increase of pulse rate had enlargement. If enlargement of the cervical glands is a symptom of Sleeping Sickness, it is possibly one which develops before the pulse rate is influenced.

It will be seen on reference to the second map, "Revised Distribution of Sleeping Sickness," that the western limit of the infective area is at the Katonga River. During my investigation a remarkable fact came to light with regard to the inhabitants of the country beyond that limit, namely, that south of this river the proportion of enlarged cervical glands lessened almost as abruptly as the proportion of *F. perstans* lessens beyond the Busoga-Kavirondo boundary. Instead of enlarged cervicals, 80 to 90 per cent. of the inhabitants in the districts south of the Katonga, namely, Buddu, Sese, Ankole, and

German East Africa, had enlarged parotids, and often submaxillary and sublingual as well. At some places, noticeably at Masaka in Buddu, it was difficult to find a native without a considerable swelling at the angle of the jaw, on both sides alike, as in mumps, a disease said by the medical officers to be common in Uganda, although I did not meet with it; in no case of enlarged glands at the angle of the jaw did I find a temperature above normal, although I examined many. (See Map "Distribution of Enlarged Glands.")

I further discovered that there existed in Buddu, in the heart of Africa, a focus of genuine bubonic plague, as Koch some years ago pointed out was the case in German East Africa.* Annually, small epidemics of very virulent bubonic plague occur in Buddu, causing the death of many natives, sometimes even of a whole village. In 1901, after a heavy mortality amongst the natives of a large missionary station, and the death of one of the White Fathers, the station was closed. Subsequently, it broke out at Fort Bukoba, in German East Africa, resulting in its temporary evacuation.

The symptoms of plague in this area of enlarged parotids, as far as I could gather, are, judging from experience in Bombay, similar to those of plague in India, with one suggestive exception, namely, buboes beneath and at the angle of the jaw are common.

I hope to be able to present to the Society further details with regard to the distribution of enlarged glands, in a subsequent report dealing with my work on the description and distribution of Filariasis in Equatorial East Africa.

### 19. MORTALITY.

Sleeping Sickness is generally believed to be always fatal, but on two or three occasions I met with persons who were willing to vouch for a recovery. The Busoga Chief, Baldani, known to the Sub-Commissioner of that district since 1890, was reported in December, 1901, to be suffering from "Mgota," and when seen he and his people admitted the fact, he himself stating that all his women had left him. He was then apparently in an advanced stage, yet, when seen by the Sub-Commissioner, 6 weeks later he appeared to be in perfect health. Histories of apparent recovery such as this are not uncommon in Uganda, but I believe they are only transient recoveries, the disease remaining quiescent for some weeks or months, after which a relapse occurs and death results.

The infective area extends for a distance of over 250 miles along the borders of the Victoria Nyanza, and no possible estimate of the total mortality could be anything more than mere guess work, but I believe it has been much greater than has been generally recognised. If one travels off the main road, visiting the shambas one by one, and entering the huts of the natives, as I have done, scores of cases will be found, previously not owned to by the chief, or native guides. The disease has decimated the population of many of the islands as well as considerable areas on the mainland.

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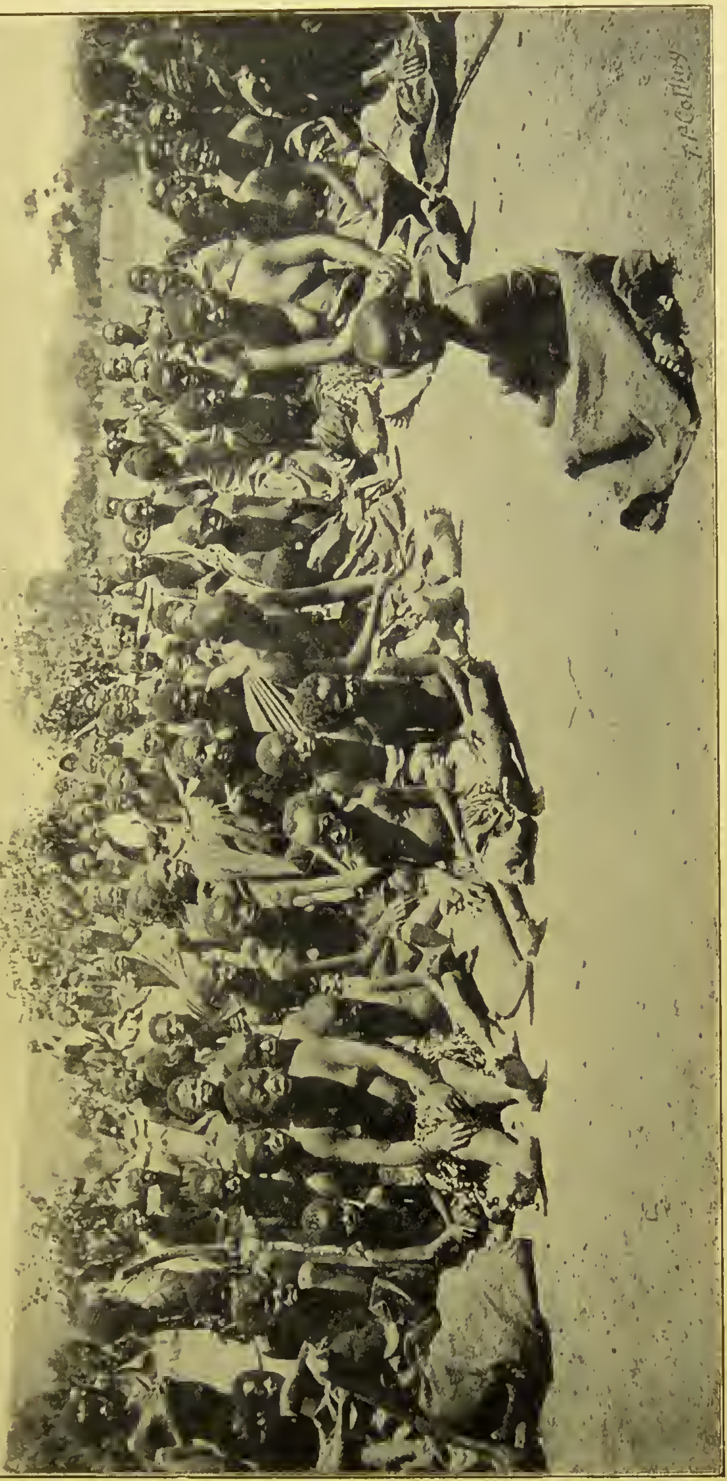
* 'Reise-Berichte über Rinderpest, Bubonenpest in Indien und Ost-Afrika, Tsetse oder Surra-Krankheit, Texasfieber tropische Malaria, Schwarzwasserfieber,' Berlin, 1898.

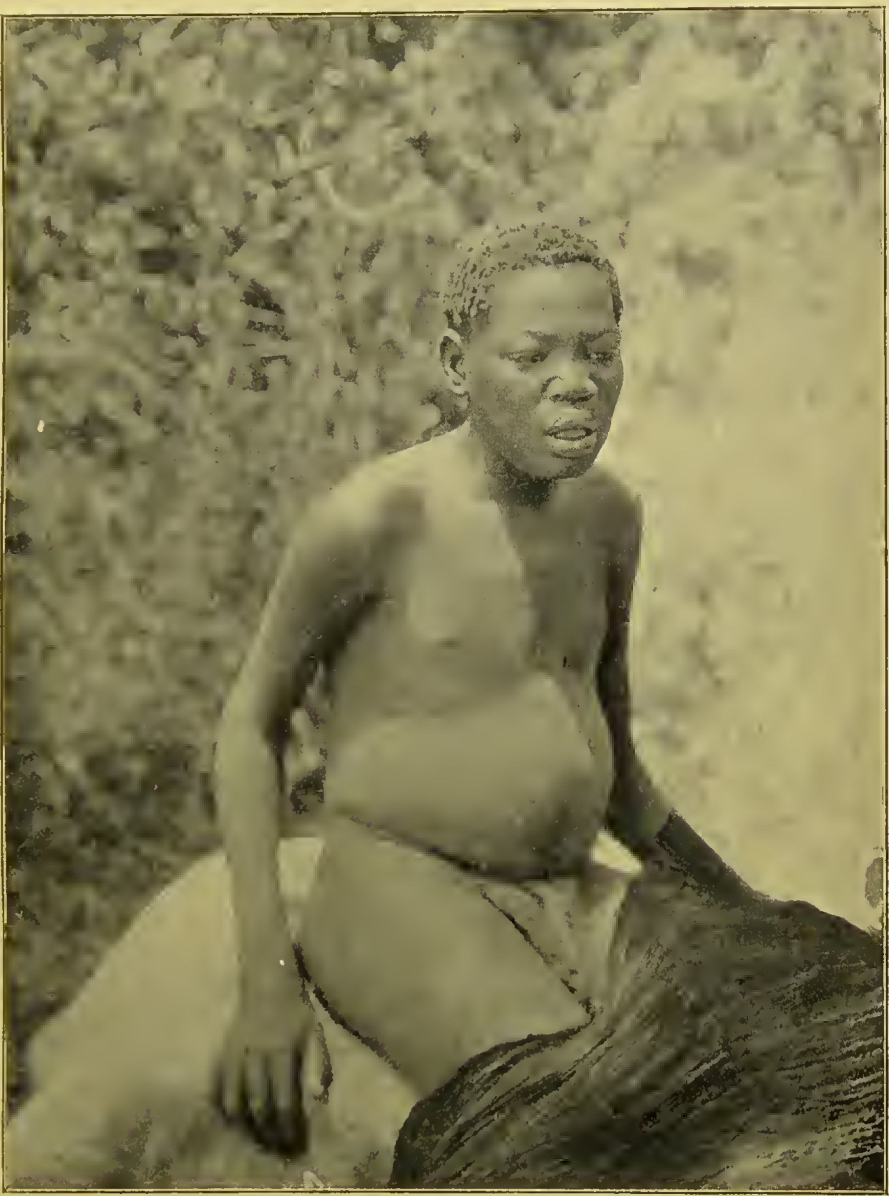
## 20. ACKNOWLEDGMENTS.

I cannot close this report without acknowledging with gratitude the help of the various officers of the Protectorate and friends, with whom I was thrown in contact in the course of my researches. Most of all I am indebted to H.M. Commissioner, Lieut.-Colonel J. Hayes Sadler, C.B., Mr. J. F. Cunningham, Chief Secretary to the Administration, Mr. George Wilson, C.B., Deputy Commissioner, Mr. W. Grant, C.M.G., Sub-Commissioner for Busoga, and the various members of the Uganda Medical Staff. I particularly wish to mention my indebtedness to the White Fathers (whose hospitality I enjoyed on many occasions) who, with their close association and knowledge of the natives, procured for me many microscope slides, and much information that I could not otherwise have obtained. I also have to thank Mr. W. G. Doggett for his valuable assistance in procuring photographs of Sleeping Sickness cases.

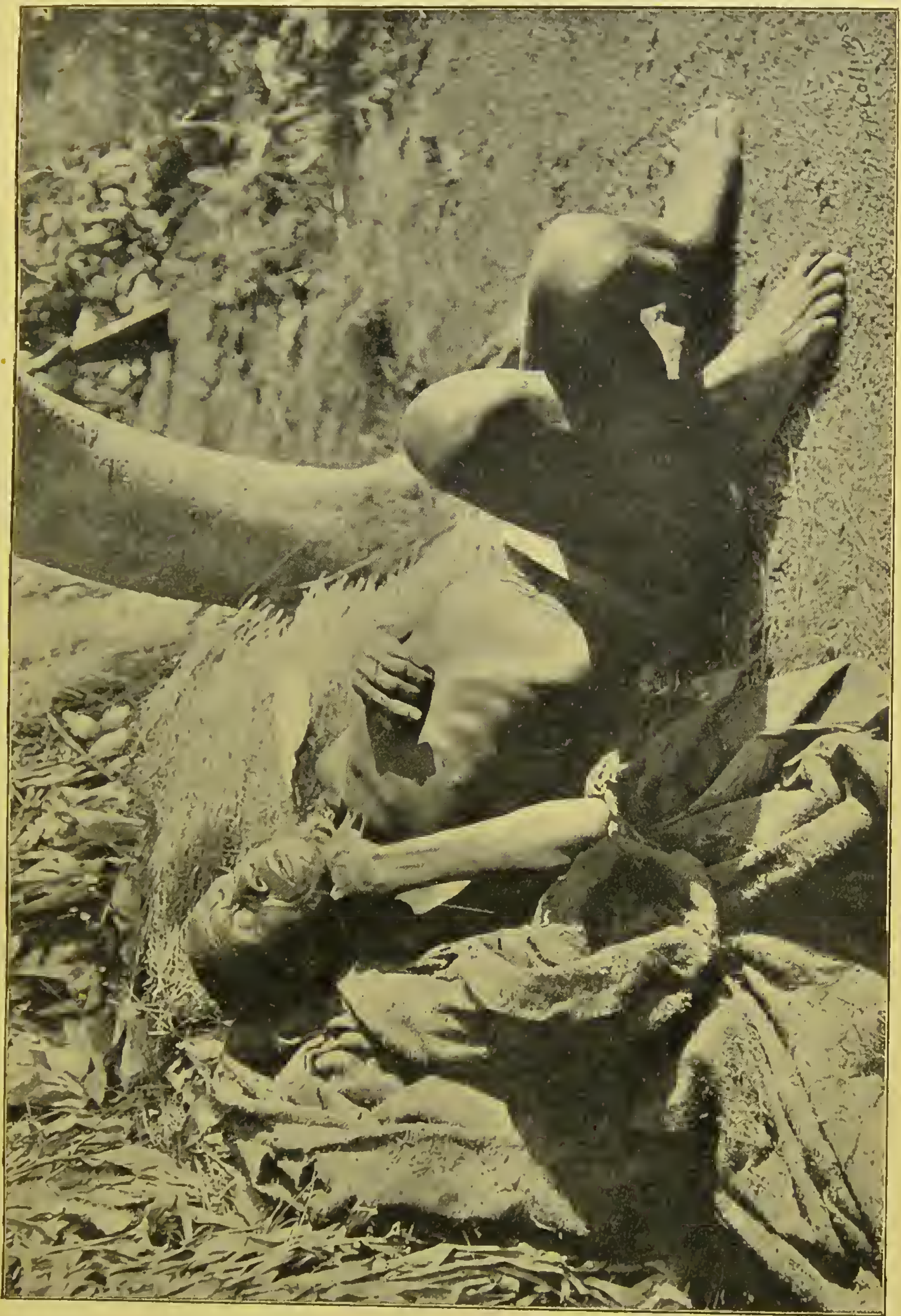
Buvuma Island. In the Shambas.



BUVUMA ISLAND. SLEEPING SICKNESS AND STARVATION.



SLEEPING SICKNESS. THE BLOATED TYPE. THE SECOND STAGE.



SLEEPING SICKNESS. LAST STAGE. LETHARGY.

# 7.

# REPORT ON A COLLECTION OF MOSQUITOES AND OTHER FLIES FROM EQUATORIAL EAST AFRICA AND THE NILE PROVINCES OF UGANDA.

(Made between June, 1902, and April, 1903.)

By FRED. V. THEOBALD, M.A., Etc.

(With Map)

The large number of Culicidæ and a few other flies collected by Dr. C. Christy during his marches while investigating the recent outbreak of Sleeping Sickness on the Victoria Nyanza are of particular interest in showing the distribution of certain species. The collection contained one new species belonging to the Culicidæ, and shows us the chief annoying forms that occur in the region traversed, the specimens having been mainly taken in tents, etc., at the different camps and not searched for otherwise. It is in these species that we should endeavour to trace out the life-history, for many of them are common in the few towns and native huts all over Central Africa. The *Tæniorhynchus fuscopennatus* seems to be very abundant judging from the bottles full brought back by Dr. Christy from so many localities. Other collectors have also sent it in numbers from Uganda.

The collection for descriptive purposes is valueless; as time was important the specimens were simply put in numbers in glass tubes and in consequence were all more or less damaged, but merely for the purpose of distribution this method of collecting is ample.

The number of species of Culicidæ identifiable was sixteen, comprising the following:—

*Pyretophorus costalis.* Loew.
*Myzomia funesta.* Giles.
*Myzorhynchus paludis.* Theobald.
*Myzorhynchus mauritianus.* Grandpre.
*Cellia pharoensis.* Theobald.
*Cellia squamosa.* Theobald.
*Christya implexa.* Nov. sp.
*Stegomyia sugens.* Wiedemann.
*Culex luteolateralis.* Theobald.
*Culex metallicus.* Theobald.
*Culex viridis.* Theobald.
*Culex fatigans.* Wiedemann.
*Culex* sp. ? (too damaged to describe).
*Tæniorhynchus fuscopennatus.* Theobald.
*Tæniorhynchus annettii.* Theobald.
*Mansonia uniformis.* Theobald.

Besides the Culicidæ there were species of *Psychodidæ*, a few *Tipulidæ*, a *Ceratopogon*, and a *Simulium*, amongst the Nematocerous flies; amongst the Brachycerous a single Tabanus, *T. dorsovitta* (Walker).

## FAMILY CULICIDÆ.

### GENUS CHRISTYA. *Nov. Gen.*

Allied to *Myzorhynchus* (Blanchard) but easily told by the long lateral tufts of abdominal scales.

Head clothed with long upright forked scales and some short scales rather broadened in front; palpi densely scaled; thorax with hair-like curved scales and narrow-curved lateral ones; prothoracic lobes with narrow-curved scales; abdomen with hairs and dense lateral tufts of long hair-like scales, the tufts apical and also other long lateral hairs; wings with dense short lanceolate lateral vein-scales; fork-cells rather short. The ♀ only known.

This genus comes in the *Anophelina* and can at once be distinguished by the marked lateral abdominal tufts of long hair-like scales. It was first taken by Dr. Christy, after whom the genus is named.

### *Christya implexa.* N. sp.

Thorax rich umber brown with golden hair-like scales and a few narrow-curved ones laterally, a narrow cinerous line on each side; pleuræ deep brown with three diverging grey lines and some small white scales; head with black and golden upright forked scales and some short golden ones in front between the eyes; palpi densely scaled with brown and white bands. Abdomen black with pale brown hairs and with long dense lateral tufts of black and golden hairs. Legs deep brown, the femora and tibiæ with white spots and to some extent the hind metatarsi; the fore and mid tarsi with the two first joints with basal white bands and to some extent the other two; in the hind legs, the apex of the metatarsi white, the apical two-thirds of the first tarsal and all the rest, except the apex of the leg, black; wings with dense blackish brown scales, the costa with two very large yellow areas, one basal and two small ones nearer the apex, the smallest the second from the apex; veins mostly dark scaled, a few yellow areas.

♀ Head black with long black upright forked scales behind, golden ones in front, and short golden ones between the eyes; palpi densely scaled with black and bands of white scales: some yellow ones at the base (damaged); proboscis black; antennæ banded black and testaceous, the basal joint testaceous, the second with dense black scales. Thorax rich dark chestnut brown with curved hair-like golden scales, small flat pale creamy ones laterally and in front, a pale grey narrow line on each side of the mesonotum due partly to the pale flat scales, partly to the grey sheen on the integument; prothoracic lobes deep testaceous with small flat creamy scales; scutellum deep brown with curved hair-like golden scales; metanotum deep brown; pleuræ deep brown with some flat white scales and with frosty grey lines, three prominent ones radiating from a central point.

Abdomen steely black with short pale brown hairs over the dorsum and with long black and golden lateral hairs, the black in prominent apical tufts; on the venter are several prominent silvery white spots forming a lateral line.

Fore legs deep brown, the femora and tibiæ with white spots, a few only on the femora and one prominent pale band, the tibiæ with six white spots, the metatarsi and first three tarsals with narrow white basal bands; mid legs as in the fore, but the femora with numerous white spots; hind legs with the femoral and tibial spots more numerous; the metatarsus much longer than the tibia, black with a narrow white apical band, the apical two-thirds of the first tarsal white, remainder white except the apical part of the foot.

Wings with dense blunt lanceolate scales, mostly brown, but some forming yellow spots; the costal border with a large yellow apical spot which spreads on to the first long vein, the next costal spot about the same size spreading on to the first long vein, but with a small median dark spot on the latter, the next costal spot minute, not spreading on to the first long vein, apical spot larger and spreading evenly on to the first long vein; veins all dark scaled otherwise, except at the base of the fork-cells and at the posterior cross-vein and a small area at the base of the third vein; fork-cells short, the first submarginal longer and narrower than the second posterior, its base nearer the base of the wing, its stem about as long as the cell, stem of the second posterior longer than the cell; fringe black, a yellow spot at the junction of the upper branch of the fifth and the lower branch of the second posterior cell; (apex ?).

*Length:* 6 mm.

*Time of capture:* August (Dr. Christy), June (Dr. Hodges).

*Habitat:* Togo (Busoga), Pokino (Toro), Mumia's boma (Kavirondo), Bulema (Ankole) (Dr. Christy), and Jinja (Busoga) (Dr. Hodges).

*Observations.*—Described from a damaged ♀ taken by Dr. Christy in August, 1902, and the description completed from a specimen taken by Dr. Hodges in June, 1903.

It cannot be confused with any other Anopheline on account of its marked abdominal character, the long lateral tufts of hair-like scales.

### *Pyretophorus costalis.* Loew.

(Mono. 'Culicidæ,' vol. 1, p. 157, and vol. 3, p. 74.)

This common African Anopheline was taken at Kamuli, the capital of the Gabula country (Busoga), in a tent on August 22; also at Sambwa, in the Bukedi country, "where mosquitoes are terrible"; also at Bikira (Buddu). The specimens are quite typical.

It has also been sent from Lagos (Nigeria), Entebbe (Uganda), Maniumba (Busoga), Togo, Cameroon, Gambia, Freetown, Bonny, Salisbury, Senegal.

### *Myzomyia funesta.* Giles.

(Mono. 'Culicidæ,' vol. 1, p. 178, and vol. 3, p. 34.)

Typical *funesta* were taken at Kamuli (Busoga), in tent with *costalis* in August; at Ngo, Gabula's country (Busoga), on the border of Lake Kioga, in August. "At Ngo, near the lake, mosquitoes were more troublesome than elsewhere in Busoga"; at Sambwa (Bukedi) with *costalis*; at Ngola Fort (Bukedi), in tent (*vide M. uniformis*); Lusinga Island (Kavirondo); and at Wadelai (Nile Provinces).

This has also been sent from Senegal, Lagos, Gambia, Natal, British Central Africa, Sierra Leone, Mashonaland, Zomba, Lake Chilwa, B.C.A., Tanganika Plateau, on Zambesi and Congo watersheds, and up the Zambesi as far as the Lupata Gorge.

### *Myzorhynchus paludis.* Theobald.

(Mono. 'Culicidæ,' vol. 1, p. 128, and vol. 3, p. 86.)

A single ♀ at Sambwa (Bukedi) with *costalis*, and another from Kisimbika (Uganda). The wings are very dark.

The same species is recorded from Kalmiga, Sierra Leone, Bahr el Ghazal.

### *Myzorhynchus mauritianus.* Grandpre.

(Mono. 'Culicidæ,' vol. 1, p. 129.)

A single ♀ at Bujaju (Buddu), and another between Mbarara and Kichwamba (Ankole).

Also recorded from Natal, Pretoria, Bahr el Ghazal, Wadi Nutrun (L. Egypt), and Mauritius.

### *Cellia pharoensis.* Theobald.

(Mono. 'Culicidæ,' vol. 1, p. 169, and vol. 3, p. 109.)

Specimens collected at Wadelai (Nile Provinces), and at Sircaos on the banks of the Nile, four marches north of Wadelai. Of the latter locality, Dr. Christy says: "The tent roof was black with this species in the morning, but none were to be seen in the evening. I noticed this same peculiarity in the case of *Cellia squamosa.* Both species adopt the 'Anopheline' attitude when at rest."

Also recorded from Gambia, Mashonaland, Rosaires, on the Blue Nile, and Cairo.

### *Cellia squamosa.* Theobald.

(Mono. 'Culicidæ,' vol. 1, p. 167, and vol. 3, p. 109.)

Specimens taken at Kajira, Masawa country (West Elgon); a single ♀ also at Lusinga Island (Kavirondo).

The same species has been recorded from Pretoria, Mashonaland, British Central Africa, Uganda.

### *Stegomyia sugens.* Wiedemann.

(Mono. 'Culicidæ,' vol. 1, p. 300.)

A few ♀'s from Entebbe (Uganda). Most of the Central African specimens are very small.

Same species also from Freetown, Nubia, Mashonaland, Gambia, and Corsica, and recently from India (Christopher).

### *Culex luteolateralis.* Theobald.

(Mono. 'Culicidæ,' vol. 1, p. 71.)

This abundant West Coast and Central African species was taken at the following localities:—Sirwanga (Busoga), in August, "specimens caught in grass at back of tent; swarms of them here, but not in tent." Lusinga Island (Kavirondo); Entebbe, "caught in Commissioner's house, and in my tent." Koko, and other places on the Buddu road, Bujaju, Masaka, Kiabogo, Bikira (Buddu); Mbarara and Kichwamba (Ankole), and at Wadelai (Nile Provinces).

Also recorded from Durban, Salisbury, Straits Settlements (rare).

### *Culex viridis.* Theobald.

(Mono. 'Culicidæ,' vol. 3, p. 212.)

Kampala (Uganda) in August, "bred from larvæ in old chattie containing almost black infusion of leaves and grass; the eggs are laid in rafts." Dr. Christy found five times as many males as females bred out from these larvæ; also taken at Kukungulo's boma near western slopes of Mount Elgon; between Mbarara and Kichwamba (Ankole); and at Mongala, the most southerly station in the Sudan.

Others have recorded it from Buse (Uganda), and Gambia.

### *Culex metallicus.* Theobald.

(Mono. 'Culicidæ,' vol. 2, p. 63.)

Only two localities in which *metallicus* occurred, namely, Nisomba in the Gabula country (Busoga) in August, and at Wadelai on the Nile in February. "Caught in tent, found in evening, scarcely one to be found in the morning."

Also recorded from Bonny, and Senegal.

This Culex seems to be very local, as but few are sent in collections from Africa.

### *Culex fatigans.* Wiedemann.

(Mono. 'Culicidæ,' vol. 2, p. 151, and vol. 3, p. 225.)

Specimens found at Sirwanga (Busoga) in August, and at Sanji (Buddu).

This species is almost cosmopolitan.

### *Culex*, sp. ?

Some damaged specimens from the Yala River (Kavirondo) where mosquitoes were rare, and from Kukungulo's boma near the western slopes of Mount Elgon, also from Lusinga Island (Kavirondo) and Bujaju (Buddu) are apparently a new species, but as they are all rubbed and damaged, they cannot be described.

### *Tæniorhynchus fuscopennatus.* Theobald.

(Mono. 'Culicidæ,' vol. 3, p. 265.)

One of the most abundant of Central African species. From the great number of specimens brought back by Dr. Christy, it is evidently very plentiful in most parts of Uganda. He took it in the following places:—Namaiba (Uganda), on Jinga Road, caught in tent in August; Budaka Fort, Bangwemi district (Mount Elgon), September; Busoro, in Kavirondo, south of Mount Elgon; Mumia's to Kisumu, in Kavirondo; Nakirebe and Koko, on Buddu Road; Sese Island; Bujaju; Masaka; Kiabogo, Bikira and Sanji, in Buddu; Kifumbero, in German East Africa; between Mbarara and Kichwamba, in Ankole; along the Katwe-Toro Road; Kibero, Marengi, Tagweta, Aombo and Keganwa, in Toto; and at Lwekula's, Nabukasi, Kinyenya, Kajongola, Masevi, Bigo, Entebbe, Kalasa, Kisimbiki, Nyenge and Kisingo, in Uganda.

Dr. Christy says: "The habits of this species are in many ways peculiar. In swamp regions it bites at any time during the day, more particularly in dull or wet weather. It is the only mosquito I have met with that can be said to fly to a light. Frequently, in the Bukedi country, beyond Lake Kioga, where clouds of this species can be seen in the day-time, quite a halo of them surrounded any lamp placed in the open, the insects flying against the glass. Their bite is not painful, and produces no subsequent irritation, as far as my personal experience goes."

### *Tæniorhynchus annettii.* Theobald.

(Mono. 'Culicidæ,' vol. 2, p. 205.)

A single ♀ from Marenge (Toro). Also recorded from Old Calabar.

### *Mansonia uniformis.* Theobald.

(Mono. 'Culicidæ,' vol. 2, pp. 187 and 180, and vol. 3, p. 273.)

This common species was taken at the following places:—Nisomba (Busoga) in August; Ngo (Busoga), on the border of Lake Choga, also in August; Sambwa (Bukedi), "caught in tent"; Erosi (Bukedi), "in tent, mosquitoes terrible"; Ngola Fort (Bukedi), "also in tent, mosquitoes terrible"; Budaka Fort, Bugwemi district (Bukedi), in September; Nbowa Fort (Bukedi), 2 hours east of Budaka, in September; Kukungulo's boma, near western slopes of Mount Elgon; Sigula Island, near Port Victoria (Kavirondo); Nyaeach and Lusinga Island (Kavirondo); Entebbe (Uganda); Sese Islands; Mbarara to Kichwamba (Ankole); Wimi River (Toro); several camps in Unyoro; and at Wadelai, on the Nile.

It has also been recorded from:—Lagos, Fort Johnson; Zomba; Gambia, at Bathurst; Chiromo; Lower Shire, B.C.A.; Old Calabar; White and Blue Niles, at Kanessa on former, Rosaires on latter; Sudan; Bahr-el-Ghazal; Lagos; Touggourt, Algeria; Natal, at Pinetown Bridge; Ceylon; Dacca; Perak; Travancore, S. India.

## FAMILY PSYCHODIDÆ.

### *Phlebotomus* sp.

Several taken at Kampala (Uganda); Dr. Christy says it is very common in Uganda in water-closets. The larvæ were discovered in the same vessel as *C. viridis* were found breeding in. This Owl Midge occurs in most collections brought back from Western and Central Africa, and appears to be generally abundant.

## FAMILY CHIRONOMIDÆ.

### *Ceratopogon* sp.

A single small Ceratopogon was taken at Sirwanga's, Tabingwa's, and Kinampere, in Busoga. Dr. Christy says: "It is very common in many places, usually near habitations. This minute fly can pass through the finest mosquito netting; muslin I have not tried. It bites terribly, leaving an irritating wheal, which itches for days. It makes a sharp short peevish buzz when settling, fully as loud as a mosquito. It attacks the wrists chiefly, but is able to pass beneath a sheet, and bite the ankles and feet. Many were frequently found full of blood on turning down the bed-clothes. I have met with a similar fly, with the same habits, at Ahmednuggar (Bombay Presidency), India."

## FAMILY SIMULIDÆ.

### *Simulium damnosum.* N. sp.

Dr. Christy remarks:—"In travelling through Busoga westward, one passes abruptly, 3 or 4 miles from Jinja, on the Nile, into a 'belt' of this terrible pest, locally known as the 'Jinja fly.' This belt extends, I believe, from the shores of the Victoria Nyanza about Lubwa's in Busoga, northwards, along the right bank of the Nile, for 12 or 15 miles or more, and is perhaps 3 or 4 miles wide. In this area the flies swarm at certain seasons in millions, and are such a plague that, according to Dr. Hodges, the Medical Officer for Busoga the natives have to leave their shambas (plantations). I do not remember to have seen the fly on the left bank of the Nile, and I cannot recollect to have met with it in any other part of the Protectorate. There is a patch of particularly dense forest within the 'Jinja fly' belt, where the flies are particularly numerous, and in passing through it I was surprised to find the fresh tracks of buffalo, now a rare animal in Uganda. The hide of a buffalo is a particularly thick one, which probably explains the reason why the animal is able to roam through belts of tsetse and other biting flies, for it has frequently been associated with the former. The bite of this small fly is a very severe one, and causes a wheal which itches intolerably, and is marked by a large drop of blood. If many flies are allowed to bite and gorge themselves the part streams with blood from the oozing of the punctures. On nearing the sphere of influence of the 'Jinja fly' each porter stops and breaks off a leafy branch to use as a fly swish, the whole of them then collect into a bunch, and travel at increased speed."

The fly is 3 mm. long, of a general black colour, head, palpi, and antennæ black except at their base, where they are bright testaceous; thorax black, the meso-thorax with bright deep golden, thick, short, curved hairs closely applied to the surface of the thorax. Meta-thorax, black, abdomen black, shiny with short black hairs, fore and mid legs black; the metatarsi of the hind legs with a broad median pale yellow band; wings bright, testaceous at the base; halteres ochraceous; the metatarsus and especially the first two tarsi much swollen in the fore legs, the two last tarsi small, very hairy, less so on the mid and hind legs; ungues all dentate.

## FAMILY TABANIDÆ.

A single species of Tabanus, the *Tabanus dorsovitta* of Walker, was taken at Bikira (Buddu). This "gad-fly," known as the "Serut Fly," occurs, not only on the West Coast, but in Central Africa and down nearly to the Sudan, for specimens have been sent me by Veterinary Colonel Griffiths, D.S.O., from Fashoda, where it is very annoying to camels and mules as well as man.

Since the identification of the insects in the collection, Dr. Christy has made a study of the distribution of each species with regard to its possible connection with either Sleeping Sickness or *Filaria perstans*, and he sends me the following notes:—

"There seems to be no species of mosquito whose distribution in any way corresponds with that of Sleeping Sickness. At no place within the infective area of that disease on the Victoria Nyanza were mosquitoes of any species a pest at the time of my visits, from July to October. Most of the country bordering the north shores of the lake is not suitable for them, being high ground, and areas of swamp are few.

"With regard to *Filaria perstans* I find that only in the case of one species does the area of distribution seem to partly correspond with that of the blood worm. This species is *Tæniorhynchus fuscopennatus*.

"Beyond the Nzoia River (Kavirondo), which is the extreme easterly limit of *F. perstans*, I only obtained this species at one locality, namely near Kisumu. With this exception the agreement of the two areas is merely a geographical one (*vide* Map).

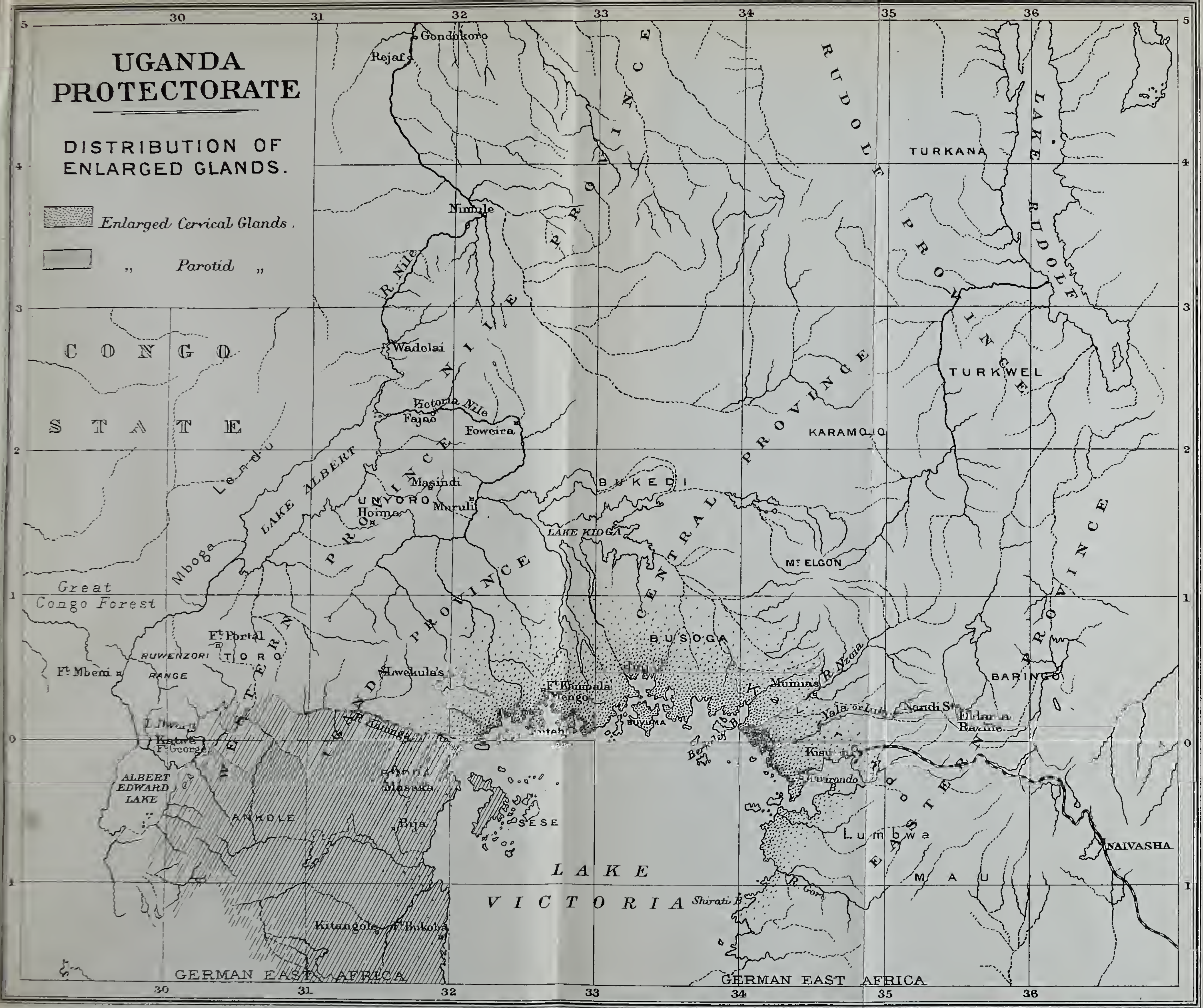
"*T. fuscopennatus* was extremely common in the province of Uganda, west of Kampala, where a high percentage of *F. perstans* existed in the blood of the general population; but on the other hand, although the species was collected in the Sese Islands, mosquitoes there were not troublesome, and the White Fathers told me that at no time in the year were they numerous; yet one of the highest recorded percentages of *F. perstans* was met with in that group of islands. In the province of Busoga also, where a high percentage of *F. perstans* was found, *T. fuscopennatus* was rare. Again, at three spots in the Protectorate the majority of the inhabitants harboured enormous numbers of a variety of *F. perstans* in their blood, yet at these places *T. fuscopennatus* was not more abundant than elsewhere, and at one of the places, namely on the slopes of Mount Elgon, I found no mosquitoes at all.

"With regard to the Tabanidæ, I met with at least two species. The genus is common in many parts of the Protectorate far away from the Sleeping Sickness area. One species (*T. dorsovitta*) was particularly abundant in the Nile Provinces. Another species belonging, I believe, to this genus, and peculiar, as far as my observations went, to Lake

Kioga, was conspicuous by its very large size, and was, I think, instrumental in the death of a mule I had with me. Unfortunately a tube containing a number of these flies, and a few Tsetse, has been lost."

Dr. Christy also sends me the following table showing the localities, grouped under Provinces, in which were collected specimens belonging to the *Anophelinæ*. The localities, he says, correspond with the different foci of malaria in regions traversed by him, and in fact include all the malarious parts of the Uganda Protectorate. The table is interesting as tending to point to the species mainly implicated in the transmission of the disease.

| Province. | Locality. | Species. | Malaria. |
|---|---|---|---|
| Kavirondo | Mumia's | *Chry. implexa* | Simple tertian. |
| | Majira (Wapoto) | *Cellia squamosa* | ? |
| | Lusinga Island | *M. funesta* | Simple tertian. |
| Busoga | Siriwanga | ? (too damaged) | ,, |
| | Naniumia | *P. costalis* | ,, |
| | Labingwa's | ? (too damaged) | ,, |
| | Gabula's (several camps) | *P. costalis* | |
| | | *M. funesta* | |
| | | *Chry. implexa* | |
| Bukedi | Sambwa (Lake Kioga) | *P. costalis* | ,, |
| | | *M. funesta* | |
| | | *M. paludis* | |
| | Ngola | *M. funesta* | ,, |
| Toro | Pokino forest | *Chry. implexa* | ,, |
| Uganda | Kisimbika | *M. paludis* | ,, |
| | Entebbe | *P. costalis* | ,, |
| Buddu | Bikiri (mission station) | *P. costalis* | ,, |
| | Bujaju | *M. mauritianus* | ? |
| Ankole | Mlema (Kagera river) | *Chry. implexa* | ? |
| Nile provinces | Wadelai and Sircows | *Cellia pharoensis* | Simple tertian and sub-tertian. |
| | Blue Nile | *Cellia pharoensis* | ,, |
| | Bahr el Ghazal | *M. paludis* | ,, |
| | | *M. mauritianus* | ,, |



Scale 1: 4,000,000 or 63·13 Miles 1 Inch
0 10 20 40 60 80 100 Miles

C. Christy.

# ROYAL SOCIETY.

# REPORTS
OF THE
# SLEEPING SICKNESS COMMISSION.

## No. IV.

8. Further Report on Sleeping Sickness in Uganda. By Lieut.-Col. David Bruce, R.A.M.C., F.R.S., David Nabarro, M.D., and Capt. E. D. W. Greig, I.M.S.

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# 8.

# FURTHER REPORT ON SLEEPING SICKNESS IN UGANDA.

By Lieut.-Col. DAVID BRUCE, R.A.M.C., F.R.S., DAVID NABARRO, M.D., and Capt. E. D. W. GREIG, I.M.S.

Received September 30, 1903.

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### *Introductory.*

Lieut.-Col. D. Bruce, R.A.M.C., and Dr. Nabarro, arrived in Entebbe, Uganda, on March 16, 1903, for the purpose of continuing the investigation of Sleeping Sickness, begun by Drs. Low, Castellani, and Christy in 1902.

On May 29, 1903, a report entitled "Progress Report on Sleeping Sickness in Uganda," was sent to the Royal Society. This was received on June 29, 1903, and published as No. 2 in the series entitled "Reports of Sleeping Sickness Commission."

Bruce and Nabarro were joined on May 25, 1903, by Capt. Greig, I.M.S., who was sent to Uganda, to study Sleeping Sickness, on behalf of the Indian Government, so that, since that date, the Commission has had the benefit of his energetic co-operation.

Lieut.-Col. Bruce left Entebbe for England on August 28, leaving Greig and Nabarro to continue the investigation.

This Further Report brings the account of the work done by the Commission up to the date of Col. Bruce's departure, and, in order to make it more easily understood, and to prevent the necessity of constant reference to the previous Progress Report, in certain places not very numerous, extracts from the latter have been incorporated into the present report.

For this, and for the mode of presentation of both reports, Lieut.-Col. Bruce is responsible.

Since this further report contains a good deal of detail, which may make it difficult to pick out easily the salient points, it may be well to point out at the outset that this report brings forward evidence to show—

1. That Sleeping Sickness is caused by the entrance into the blood and cerebro-spinal fluid of a species of trypanosoma.

2. That this species is probably that discovered by Forde and described by Dutton from the West Coast of Africa, and called by him *Trypanosoma Gambiense*.

3. That the so-called cases of trypanosoma fever, described from the West Coast, may be and probably are, cases of Sleeping Sickness in the earliest stages.

4. That monkeys are susceptible to Sleeping Sickness, and show the same symptoms, and run the same course, whether the trypanosomes injected are derived from cases of so-called trypanosoma fever, or from the cerebro-spinal fluid of cases of *Sleeping Sickness*.

5. That dogs and rats are partially susceptible, but that guinea-pigs, donkeys, oxen, goats, and sheep, up to the present, have shown themselves absolutely refractory.

6. That the trypanosomes are transmitted from the sick to the healthy by a species of tsetse fly, *Glossina palpalis*, and by it alone.

7. That the distribution of Sleeping Sickness and *Glossina palpalis* correspond.

8. That Sleeping Sickness is, in short, a human tsetse fly disease.

For the sake of the future historian, it may be well to point out the exact state of affairs as regards our knowledge of Sleeping Sickness in relation to trypanosomes, when Col. Bruce and Dr. Nabarro arrived in Entebbe, on March 16, 1903. Dr. Castellani had observed these hæmatozoa in the cerebro-spinal fluid of five cases of Sleeping Sickness, and in one of these he had also seen them in the blood.

At the time of the arrival of the Commission, he did not consider that this trypanosoma had any causal relationship to the disease, but thought that it was an accidental concomitant like *Filaria perstans*. When he reported his observation to Lieut.-Col. Bruce, the latter was much struck by the discovery, and urged Dr. Castellani to pursue this point during his few remaining days in Entebbe.

Dr. Moffat, the Principal Medical Officer of Uganda, Dr. Baker, the Resident Medical Officer at Entebbe, as well as Colonel and Mrs. Bruce worked from morning to night in co-operation with Dr. Castellani, in order to confirm his observation, with the result that, before he left for England on April 6, 34 cases of Sleeping Sickness and 12 controls had been examined, with a positive result in 70 per cent. of the former, and a negative result in the latter.

As Dr. Castellani has not entered into any detail respecting these matters in his reports, it is thought advisable to supplement his account with the above, as the true history of the discovery of the cause of any important disease must always be of interest.

This most interesting discovery of Dr. Castellani's, which was due to his introduction of the method of centrifuging the cerebro-spinal fluid in his search for his streptococcus, has been of the utmost possible value to the present Commission. It put them at once on the right track and led to the rapid and easy elucidation of the etiology of this hitherto mysterious disease. Without a knowledge of his observation they might have worked for months in the dark, and, in truth, they might even have returned to England still ignorant as to the true cause of the disease.

After Dr. Castellani's departure for England, the Commission continued this line of work, with the result that the cerebro-spinal fluid of every case of Sleeping Sickness examined was found to contain trypanosomes.

The members of the Commission take the earliest opportunity of expressing their heartiest thanks to Colonel Hayes-Sadler, C.B., His Majesty's Commissioner and Consul-General, Uganda, for his invariable kindness and good offices, to Dr. Moffat, C.M.G., who has been untiring in help, to Dr. Hodges, Acting Principal Medical Officer, during Dr. Moffat's absence, to Dr. Baker, the resident colonial surgeon in Entebbe, who has spent much of his leisure time in working with and for the Commission, to Dr. Wiggins, of Kisumu, who has given valuable assistance, to the sub-commissioners, collectors, and other officials of the Protectorate, to the bishops of the various churches and their missionaries, and lastly to the Prime Minister and Regents of Uganda, who are now helping to make out the distribution of tsetse flies in

Uganda, and have throughout shown great interest in the work. Our best thanks are also due to Professor Ray Lankester, the Director, and to Mr. Austen, the Entomologist of the British Museum for their kindness in naming the many species of biting flies sent to them from Uganda. In Mr. Austen's recent monograph of the tsetse flies, a full account of *Glossina palpalis* is given.

### 1. *The Cerebro-Spinal Fluid of Every Case of Sleeping Sickness taken by Lumbar Puncture during Life contains Trypanosomes.*

In the Progress Report, it was shown that, in every case of Sleeping Sickness, trypanosomes are found in the cerebro-spinal fluid, and a table giving in detail the examination of forty cases, was given.

It was also pointed out that the trypanosomes exist, as a rule, in very small numbers, and it may be considered impossible that such small numbers can bring about pathological changes, and be the cause of such a grave disease as Sleeping Sickness. If, however, we turn to another trypanosoma disease, Nagana in the native goat or sheep, we find the same scarcity of parasites. It is only rarely that the trypanosomes can be detected in the blood, and yet the disease invariably pursues its chronic course to a fatal termination.

### 2. *Trypanosomes occur in the Cerebro-Spinal Fluid of Cases of Sleeping Sickness in other Districts than Uganda.*

It will be remembered that almost all cases of Sleeping Sickness examined in Entebbe, show the presence in the blood of *Filaria perstans*, whereas blood examinations of cases of the same disease in Kavirondo, British East Africa, practically show no Filaria.

In the same way it may be that every case of Sleeping Sickness admitted to the Entebbe Hospital, from the surrounding district, may harbour trypanosomes, while cases occurring in Kavirondo may be free from them.

Dr. Wiggins who is stationed at Kisumu in Kavirondo, and is in charge of a Sleeping Sickness Hospital there, kindly undertook to examine cases for the Commission. He had been on leave in Entebbe for a fortnight in April, and had then been most energetic in examining cases, and hence was familiar with the methods used in demonstrating the parasites.

On May 25, he sent in his results which are as follows:—

Sleeping Sickness—

| | | | |
|---|---|---|---|
| 1st stage, | 31 cases. | Trypanosomes present in | 31. |
| 2nd ,, | 15 ,, | ,, ,, | 15. |
| 3rd ,, | 7 ,, | ,, ,, | 7. |

Dr. Wiggins is of opinion that the trypanosomes are much more numerous in the cases in Kavirondo than in Entebbe. If this is so, it is a curious fact, but until we have some more exact method of numeration, the observation must remain not proven.

The trypanosoma then is present in the cerebro-spinal fluid of all cases of Sleeping Sickness in Kavirondo, as well as in Uganda, and this, which seemed to us an important question, has been satisfactorily answered.

### 3. *The Cerebro-spinal Fluid taken by Lumbar Puncture from Cases of Disease other than Sleeping Sickness do not contain Trypanosomes.*

In the Progress Report, a table was given showing that trypanosomes were absent from the cerebro-spinal fluid of 15 cases admitted to hospital for various diseases other than Sleeping Sickness. Dr. Wiggins also writes that he has examined 7 cases of disease other than Sleeping Sickness at Kisumu, and failed to find trypanosomes in any of them.

From the above table and Dr. Wiggins' report, although the numbers are small, it would appear that the cerebro-spinal fluid taken by lumbar puncture during life from cases of disease other than Sleeping Sickness does not contain trypanosomes.

### 4. *The Peripheral Blood of all Cases of Sleeping Sickness also contains the Trypanosomes.*

This point was also dealt with in the progress report, with the result that it was shown that practically all cases of Sleeping Sickness harbour trypanosomes in the blood as well as in the cerebro-spinal fluid.

### 5. *Has the so-called Trypanosoma Fever any Connection with Sleeping Sickness?*

On the West Coast of Africa, during the past few years, trypanosomes have been found in the blood of several persons, and the name trypanosoma fever has been given to that disease. The parasites have not been confined to the blood of natives, but at least three white people have harboured them. No suspicion of these being cases of Sleeping Sickness has been entertained by those in medical charge of them. The symptoms are different. Trypanosoma fever appears to cause little or no inconvenience to those harbouring the parasite, whereas in Sleeping Sickness the disease is a well-marked one and is invariably fatal. Can there be any connection between the two?

In March last, Dr. Baker of the Colonial Service, who is stationed here, discovered trypanosomes in the blood of five natives, and

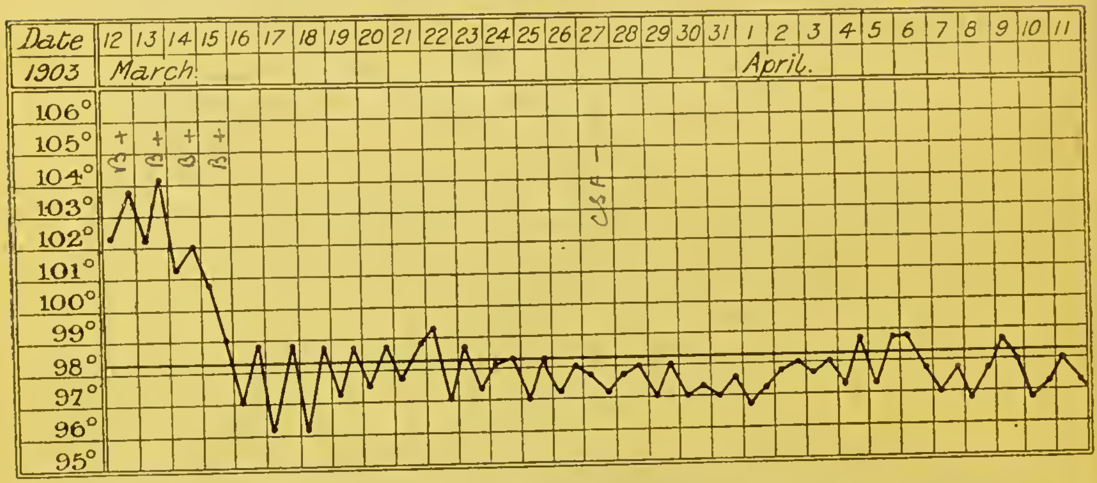
Dr. Moffat, the Principal Medical Officer, has also found them in the blood of a European.

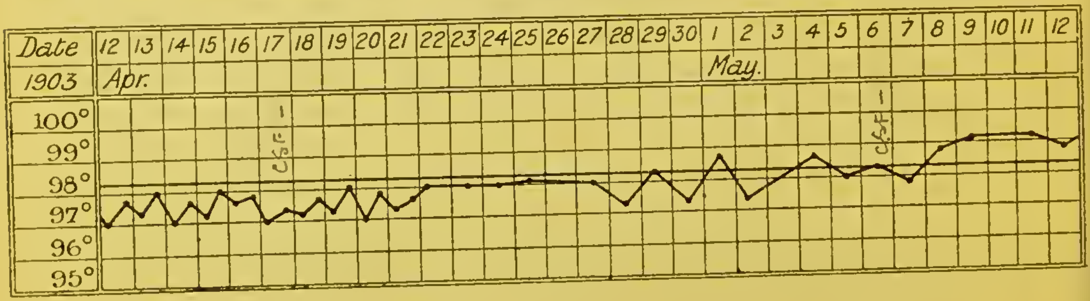
As we are face to face with a curious problem, we will with the permission of Drs. Moffat and Baker, give a temperature chart and a short description of each of these cases. The subject is an interesting and comparatively new one, and it will be seen that there is probably a close connection between the two diseases.

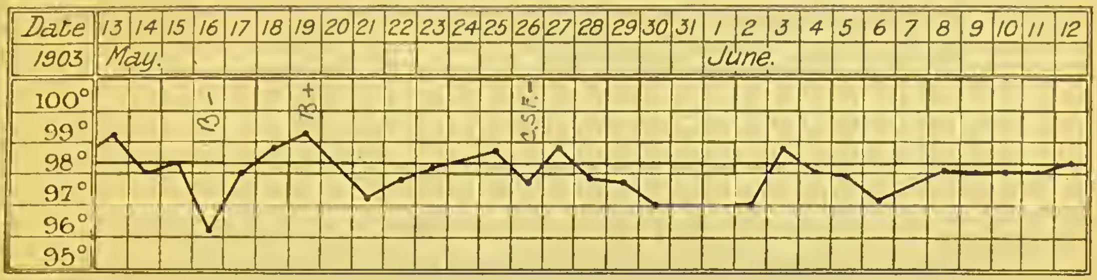
CASE 31.—Karala Barigi. Male. District, Singo. Occupation, Policeman. Tribe, Mundu, Nubian.

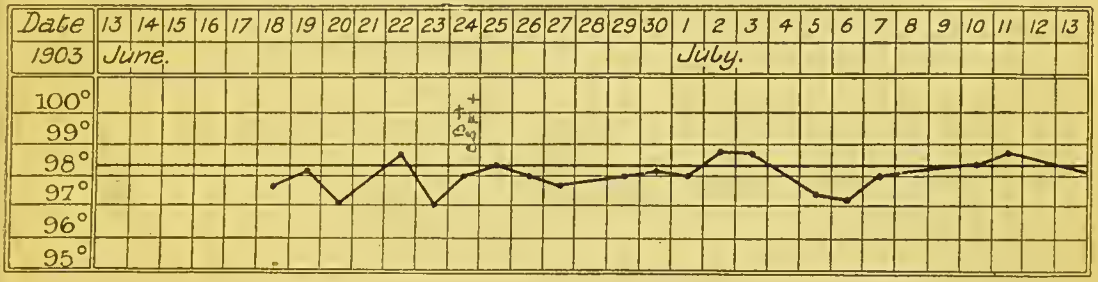
April 24, 1903. Patient states that he has been 6 months in Entebbe. His illness began on March 10. He asserts that at present he feels quite well, and has no headache or other symptom. There are no enlarged glands in the neck, but in the axilla they are as large as peas, and they are also enlarged in the groin. His tongue is moist and furred. His speech is fluent. Pulse 120, fair. His heart sounds are normal.

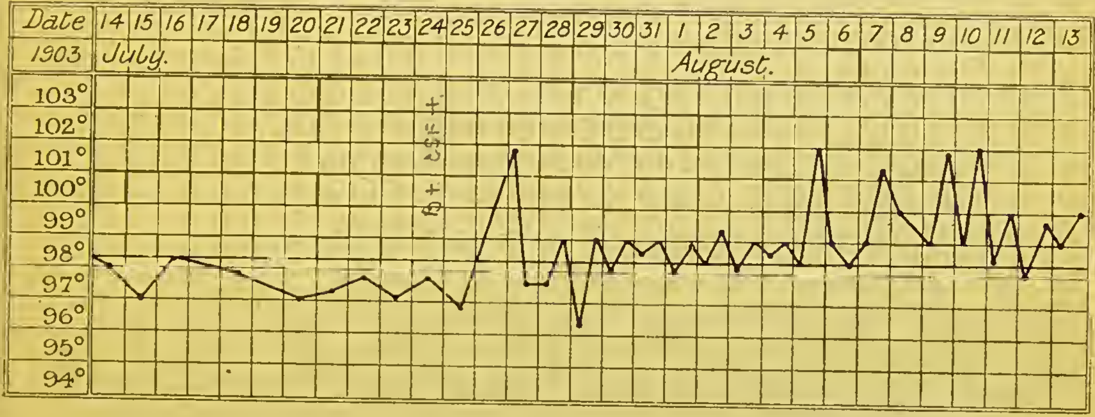
The following chart represents the temperature curve :—






Date
1903
13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12
May.
June.
100°
99°
98°
97°
96°
95°
B –
B +
C.S.F. –


Date
1903
13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13
June.
July.
100°
99°
98°
97°
96°
95°
B +
C.S.F. +


Date
1903
14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13
July.
August.
103°
102°
101°
100°
99°
98°
97°
96°
95°
94°
B + C.S.F. +

The following table shows the presence or absence of trypanosomes in the blood and cerebro-spinal fluid:—

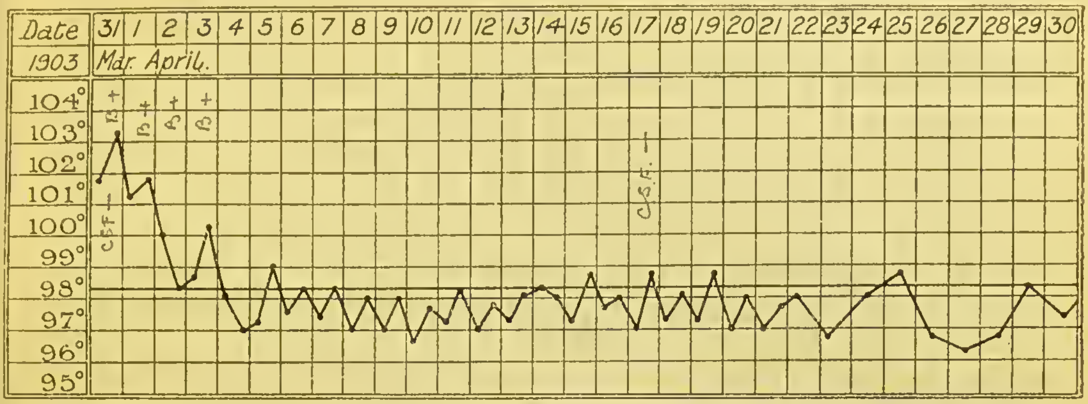
| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 12 . . . . | | | + | | |
| ,, 13 . . . . | | | + | | |
| ,, 14 . . . . | | | + | | |
| ,, 15 . . . . | | | + | | |
| ,, 27 . . . . | | | | − | − |
| Apr. 17 . . . . | | | | − | − |
| May 6 . . . . | | | | − | − |
| ,, 16 . . . . | + | | − | | |
| ,, 19 . . . . | | | + | | |
| ,, 26 . . . . | | | | − | − |
| June 24 . . . . | + | | + | | + |
| July 24 . . . . | | | + | | + |

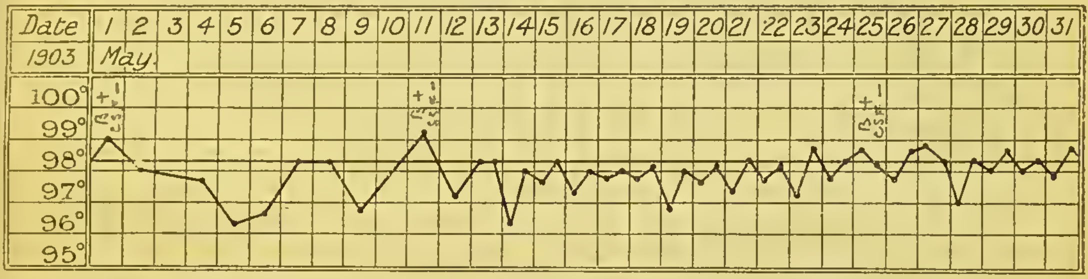
*Remarks.*—This is an important and suggestive case. Karala Barigi was admitted to hospital on March 12, and his disease was diagnosed trypanosoma fever. After a few days his temperature becomes normal, and he remains apparently in good health, and does his duty as a policeman until the end of July, when his temperature again becomes unsettled, and he is again admitted into hospital. During all this time the trypanosomes have been found in his blood, but it is only towards the end of June that they are found in the cerebro-spinal fluid, and this corresponds fairly well with the rise in temperature. The temperature curve is also very suggestive of Sleeping Sickness, and it is suggested that this case of trypanosoma fever is now passing on to Sleeping Sickness. At the same time it must be admitted that up to the present date he shows no marked symptoms of Sleeping Sickness.

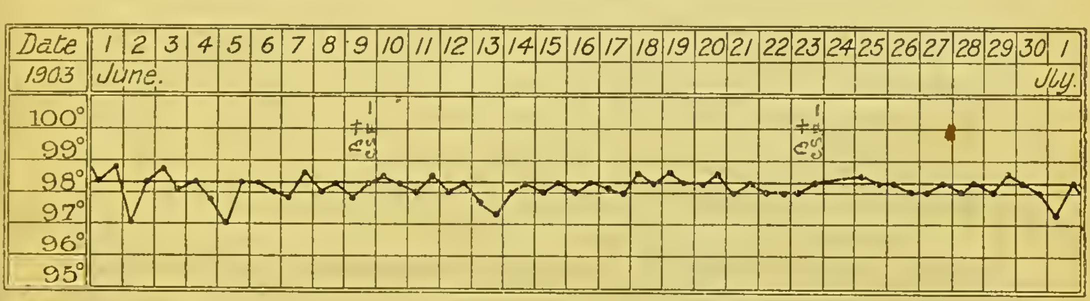
CASE 64.—Jordien Murjan. Male. Age 35 years. District, Muru. Nubian. Occupation, Prisoner, for last 2 Years.

March 31, 1903. Admitted to hospital. He is an old Soudanese mutineer, and lives as a prisoner in the jail. No fellow prisoners have had Sleeping Sickness. He has no œdema, and no noticeable swelling of glands. His tongue is healthy, but shaky. There is no tremor of the hands. His speech is normal, and pulse 144.

The following chart represents the temperature curve:—


Date
1903
31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
Mar. April.
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
CSF −
B +
B +
B +
B +
C.S.F. −


Date
1903
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
May.
100°
99°
98°
97°
96°
95°
B + CSF −
B + CSF −
B + CSF −


Date
1903
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1
June.
Jly.
100°
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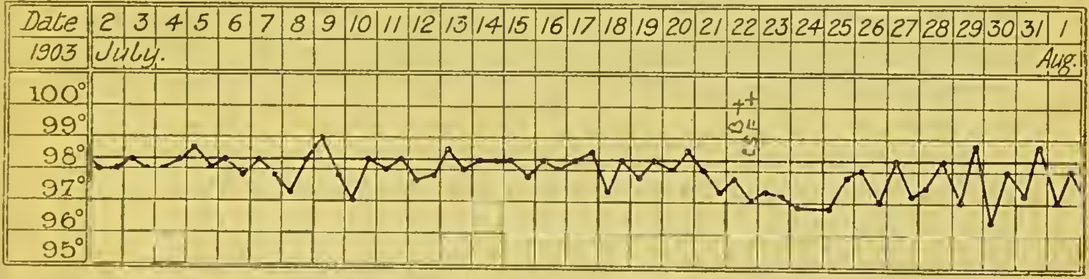
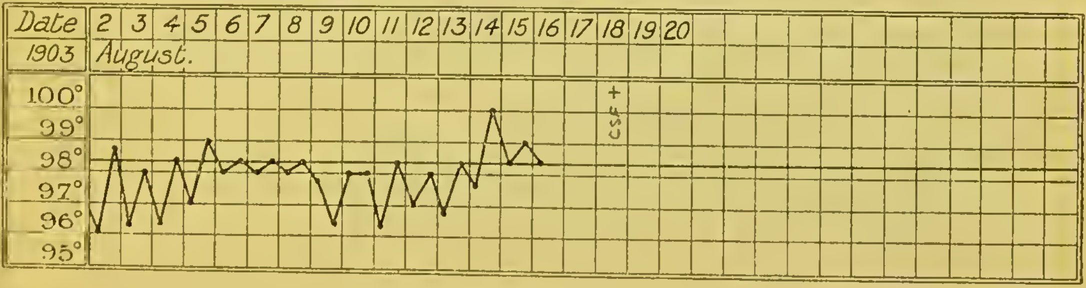
96°
95°
B + CSF −
B + CSF −


Date
1903
2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1
July.
Aug.
100°
99°
98°
97°
96°
95°
B + CSF +


Date
1903
2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
August.
100°
99°
98°
97°
96°
95°
CSF +

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 31 .... | | | + | | − |
| Apr. 1 .... | | | + | | |
| ,, 2 .... | | | + | | |
| ,, 3 .... | | | + | | |
| ,, 17 .... | | | | − | − |
| May 1 .... | − | − | + | | − |
| ,, 11 .... | | | + | | − |
| ,, 25 .... | | | + | | − |
| June 9 .... | − | | + | | − |
| ,, 23 .... | − | | + | | − |
| July 22 .... | | | + | | + |
| Aug. 18 .... | | | | | + |

*Remarks.*—Here again we have a case closely resembling that of Karala Barigi. The trypanosomes are constantly found in the blood, although the man appears to be in good health. On July 22 the trypanosomes have found their way into the cerebro-spinal fluid, and it is suggested that this case is also going on to Sleeping Sickness.

In the Progress Report the cases of Kumsarsabba, Tabula, and Bara Risgallah were also given. These men are still under observation, but nothing has happened. The trypanosomes are found regularly in their blood, but never in the cerebro-spinal fluid. Their temperature is constantly normal. The men are at duty, and say they feel strong and well.

From these cases it will be seen that the trypanosomes, when confined to the blood, do not visibly affect the health of their native hosts. The question seems to resolve itself into either the trypanosomes found in the blood, and in the cerebro-spinal fluid belong to different species, and give rise to different diseases, or they are one and the same, and if confined to the blood give rise to slight feverish symptoms, whereas if they gain entrance to the cerebro-spinal fluid they give rise to Sleeping Sickness. The evidence is not strong enough, at present, to justify a dogmatic answer to the question, but what there is points to the trypanosomes being one and the same, and trypanosoma fever the first stage of Sleeping Sickness.

### 6. *Does the Blood of the General Population contain Trypanosomes?*

On May 18 the Commission received a telegram from the Royal Society, asking for statistics regarding the prevalence of trypanosomes in the general population (*a*) inside, and (*b*) outside the Sleeping Sickness area. On a little consideration the importance of such an inquiry is seen. If the trypanosoma found in the blood of Karala Barigi and the others belongs to the same species as that found in Sleeping Sickness, then a larger percentage of the general population inside the Sleeping Sickness area should be found to harbour trypanosomes, than those living in the Non-Sleeping Sickness area. In fact, if the supposition be true, there ought not to be a single trypanosoma found in the blood of persons living in a clean area. Because, if the theory that trypanosomes in the blood is merely the first stage of Sleeping Sickness be true, then you cannot have all the cases in the Non-Sleeping Sickness area in the initial stage, but scattered among them you would naturally expect to find some in the more advanced stage.

In Entebbe, the hut-tax labourers supply ample material for such an investigation. There are some 1,500 employed here at present, and they come from all parts of the Protectorate. They remain at work in Entebbe for one month, and then return to their homes. During this time they are liable to contract Sleeping Sickness, but for the purpose of this inquiry natives from Non-Sleeping Sickness areas, who had only been a few days in Entebbe, were chosen. Capt. Greig also went over to Nairobi, in British East Africa, and examined 50 cases in an undoubted Non-Sleeping Sickness area.

The results of these examinations are given in the following lists. These lists are given in full, and will be useful six months or a year hence in order to trace out and learn the fate of the men whose blood now show trypanosomes, but who, at present, have no symptoms of Sleeping Sickness.

## *Incidence of Trypanosomes in the Blood of General Population.*

### A.—Sleeping Sickness Area.

| Date. | Name. | Age. | Sex. | District. | Shamba. | Name of chief. | Trypanosoma. | Filaria. |
|---|---|---|---|---|---|---|---|---|
| June 12 | Wagononye | 25 | M. | Sese Island | Sewaua | .. | + | + |
| ,, 13 | Zamala | 20 | M. | ,, | Kaganda Island | .. | − | + |
| | Sibolyamba | 25 | M. | .. | Semagala Island, Malanga | .. | + | + |
| | Lusaka | 30 | M. | .. | Kaganda Island, Bubeki | .. | − | + |
| | Byasi | 20 | M. | .. | Semagala Island, Busendeni | Serumaga | − | + |
| | Nalyonzi | 25 | M. | .. | Buvovu Island, Kasange | Nokuba | − | + |
| | Sagala | 25 | M. | .. | Kaganda Island, Bukasa | Kagauda | − | + |
| ,, 16 | Paulo | 20 | M. | .. | Semagala Island, Dajs | Bugambi | − | + |
| | Luwercheti | 30 | M. | .. | Buvu Island | Namamba | − | + |
| | Balyokwakwe | 35 | M. | .. | Semagala Island, Burigo | Kakobogo | − | − |
| | Kapere | 20 | M. | ,, | Semagala Island, Busowa | Sawadu | − | − |
| | Kewaza | 20 | M. | ,, | Semagala Island, Bungo | Kakobogo | − | + |
| | Takirambala | 40 | M. | ,, | Lubamba Island, Impenja | Sabawabe | − | − |
| | Gumira | 40 | M. | ,, | Bugaba Island | Bugu | + | + |
| | Kitungula | 25 | M. | ,, | Semagala Island, Buswa | Keozekia | + | − |
| | Tevamukopi | 35 | M. | ,, | Bunami Island | Buvaro | + | + |
| | Kajerero | 25 | M. | ,, | Buninga Island | Savawan | − | + |
| ,, 18 | Zwaki | 23 | M. | Kome Island | Kome Island | .. | + | + |
| | Nutaba | 24 | M. | Bugaba Island | Bugaba Island | .. | + | + |
| | Nmmugula | 25 | M. | Sewaia Island | Sewaia Island | .. | − | − |
| ,, 19 | Sibaganga | 30 | M. | Bussi Island | Bulenge | Mugema | + | − |
| ,, 23 | Tangamalala | 25 | M. | .. | Bussi Island | Rasto | + | + |
| | Nasajage | 30 | M. | .. | .. | .. | + | + |
| | Tabula | 10 | M. | .. | .. | .. | − | + |
| | Mundu | 25 | M. | .. | .. | .. | − | + |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| ,, 27 .. | Mondu | 20 | M. | ,, | ,, | Mugema | – | + |
| | Buza | 30 | M. | ,, | .. | ,, | + | + |
| | Sabakake | 20 | M. | ,, | .. | .. | + | + |
| | Bagamatuba | 30 | M. | ,, | ,, | ,, | – | + |
| | Nangibwa | 30 | M. | ,, | ,, | ,, | – | + |
| .. 8 .. | Magwanjemba | 40 | M. | Kagagara | .. | .. | + | + |
| ,, 13 .. | Rotate | 45 | M. | Nkumba | Nkumba | Sebugwa | – | + |
| | Junguberase | 25 | M. | ,, | ,, | ,, | – | – |
| | Mucase | 25 | M. | ,, | ,, | ,, | + | – |
| | Sefebedi | 40 | M. | ,, | ,, | ,, | – | – |
| | Mundu | 30 | M. | ,, | ,, | ,, | – | + |
| | Kikocharwasi | 35 | M. | ,, | ,, | ,, | – | – |
| ,, 19 .. | Petero | 20 | M. | Entebbe | Ugonga | .. | – | – |
| | Sibasoboke | 30 | M. | Bussi Island | Gombe | Mugema | – | + |
| .. 20 . | Arikisi | 40 | M. | Magema | Mugema | Sebugwao | – | + |
| | Antonio | 20 | M. | ,, | ,, | ,, | – | – |
| | Kiguaku | 30 | M. | ,, | ,, | ,, | – | – |
| | Brahmi | 25 | M. | ,, | .. | ,, | – | – |
| | Musoke | 14 | M. | ,, | .. | ,, | – | – |
| | Daniel | 12 | M. | .. | ,, | Sebanji | – | – |
| | Balumedeni | 20 | M. | ,, | ,, | ,, | – | – |
| | Semone | 20 | M. | ,, | ,, | ,, | – | + |
| | Muagazi | 30 | M. | ,, | .. | Mugema | – | + |
| | Nyasi | 12 | M. | .. | .. | Sebanja | – | – |
| .. 22 .. | Saulo | 18 | M. | .. | ,, | Mugema | + | – |
| | Zake | 20 | M. | ,, | .. | ,, | – | + |
| | Kalulwe | 19 | M. | Entebbe | Kigungu | Mugula | + | + |
| | Kakulwana | 18 | M. | ,, | ,, | Wasanye | – | + |
| .. 22 .. | Zirimanye | 18 | M. | ,, | ,, | Mugula | – | – |
| | Danielli | 16 | M. | ,, | .. | Batende | – | – |
| | Daudi | 20 | M. | ,, | 4 miles | Kalunga | – | + |
| | Bafirawara | 16 | M. | .. | Zinderi | Mugula | – | – |
| | Mutesenda | 20 | M. | ,, | 3 miles | ,, | – | – |
| | Lugwana | 18 | M. | ,, | Kabale | Sebugwao | – | – |
| | Gabula | 20 | M. | .. | .. | ,, | ∴ | – |
| March 12 .. | Karala Barigi | .. | M. | ,, | Policeman | .. | + | + |

A.—Sleeping Sickness Area—*continued.*

| Date. | Name. | Age. | Sex. | District. | Shamba. | Name of chief. | Trypano-soma. | Filaria. |
|---|---|---|---|---|---|---|---|---|
| March 28 .. | Kumsarsabba ...... | .. | M. | Entebbe ....... | Policeman............... | .. | + | — |
| ,, 31 .. | Jordien Murjan..... | .. | M. | ,, ....... | Prisoner ............... | .. | + | — |
| April 15 .. | Tabula .......... | .. | M. | ,, ....... | Marine ................ | .. | + | + |
| ,, 21 .. | Bara Risgallah ..... | .. | M. | ,, ....... | Policeman............... | .. | + | + |
| ,, 23 .. | Airara............ | .. | M. | ,, ....... | Prisoner ............... | .. | — | — |
|  | Biggity ........... | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 24 .. | Nathaniel.......... | .. | M. | ,, ....... | Hospital ............... | .. | — | — |
| ,, 28 .. | Aradiki........... | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 29 .. | Chia Msoga ........ | .. | M. | ,, ....... | ,, ............... | .. | — | — |
|  | Arkadi ...... ..... | .. | M. | ,, ....... | ,, ............... | .. | — | — |
|  | Mandandiki ........ | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| May 6 .. | Jumabini .......... | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 8 .. | Matea ............ | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 13 .. | Bifarawala ......... | .. | M. | ,, ....... | Prisoner ............... | .. | — | — |
| ,, 16 .. | Amuri Abdulla ..... | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 18 .. | Screwame .......... | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 21 .. | Juma Bin Abdulla .. | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| ,, 22 .. | Baraca Bin Salimi .. | .. | M. | ,, ....... | ,, ............... | .. | — | — |
| April 2 .. | J. M.............. | .. | M. | ,, ....... | Hospital ............... | European ...... | + | — |

## *Incidence of Trypanosomes in the Blood of General Population.*

### B.—Non-Sleeping Sickness Area, Uganda.

| Date. | Name. | Age. | Sex. | District. | Shamba. | Name of chief. | Trypanosoma. | Filaria. |
|---|---|---|---|---|---|---|---|---|
| June 2...... | Wagalla ........... | 40 | M. | Kampala ........ | .. | .. | — | + |
|  | Zemadari .......... | 20 | M. | ,, ........ | .. | .. | — | + |
|  | Mutakanya ........ | 25 | M. | ,, ........ | .. | .. | — | — |
|  | Suziga ............ | 20 | M. | ,, ........ | .. | .. | — | — |
| ,, 3...... | Burrigardi ......... | 30 | M. | Toro............ | Kaima............... | .. | — | — |
|  | Ntaragi ........... | 40 | M. | Makota .......... | 5 days N. of Kampala.. | .. | — | — |
|  | Bassaga ........... | 15 | M. | ,, ......... | ,, .. | .. | — | — |
|  | Kilongazi .......... | 50 | M. | Jalamba ........ | Near Makota ......... | .. | — | + |
| ,, 4...... | Kianju ............ | 40 | M. | Buganga ......... | .. | .. | — | — |
|  | Nsali ............. | 40 | M. | ,, ........ | .. | .. | — | — |
|  | Jagenda ........... | 35 | M. | Singo ........... | Matiana ............ | .. | — | — |
|  | Sengova............ | 30 | M. | Buganga ......... | .. | .. | — | — |
|  | Kidormori ......... | 30 | M. | Nakolia.......... | .. | .. | — | + |
|  | Bagnsagera ........ | 25 | M. | ,, .......... | .. | .. | — | — |
| ,, 5...... | Luganda ........... | 20 | M. | Kalagala ......... | .. | .. | — | — |
|  | Birlla.............. | 35 | M. | Matuntotora ..... | .. | .. | — | + |
|  | Kagema ......... .. | 45 | M. | ,, ..... | .. | .. | — | + |
|  | Kasoka ............ | 35 | M. | Kibali........... | .. | .. | — | — |
|  | Bulabiki ........... | 40 | M. | Kiwangole ....... | .. | .. | — | — |
| ,, 8...... | Kongura........... | 35 | M. | Kikanda ......... | .. | .. | — | — |
| ,, 10.. ... | Watagana ......... | 35 | M. | Bulemwezi ....... | Busibika ............ | .. | — | — |
|  | Karimuemba ....... | 40 | M. | Kalagala ......... | Kalagala ............ | .. | — | — |
|  | Watesaka .......... | 20 | M. | Bulemwezi....... | Sambwao ........... | .. | — | + |
|  | Matamburire....... | 35 | M. | ,, ....... | Busindi.......... .. | .. | — | — |
|  | Roukiaju .......... | 30 | M. | ,, ....... | Sambwao ........... | .. | — | + |
|  | Kaisi.............. | 40 | M. | ,, ....... | ,, ........... | .. | — | + |
| ,, 11 ..... | Guatude ........... | 25 | M. | Mawokota ....... | Kaniki .............. | .. | — | — |
|  | Murianzeki ........ | 30 | M. | ,, ....... | ,, .............. | .. | — | + |
|  | Tandamuwaka ...... | 30 | M. | ,, ....... | ,, .............. | .. | — | + |
|  | Banegwira ......... | 30 | M. | ,, ....... | ,, .............. | .. | — | + |
|  | Basaga............. | 30 | M. | ,, ....... | ,, .............. | .. | — | + |
|  | Muayowa .......... | 35 | M. | ,, ....... | ,, .............. | .. | — | + |

Non-Sleeping-Sickness Area, Uganda—*continued.*

| Date. | Name. | Age. | Sex. | District. | Shamba. | Name of chief. | Trypanosoma. | Filaria. |
|---|---|---|---|---|---|---|---|---|
| June 12 | Kawaratuli | 45 | M. | Buddu | Masaka | .. | − | + |
| | Kyalwazi | 45 | M. | ,, | ,, | .. | − | + |
| | Byempulidi | 40 | M. | ,, | ,, | .. | − | − |
| | Ganantawa | 40 | M. | ,, | .. | .. | − | + |
| | Sabagwera | 25 | M. | ,, | Dinnu ? | .. | − | + |
| ,, 15 | Kubolikoza | 20 | M. | Mawokota | Kaima | Rutalo | − | − |
| | Semukotu | 30 | M. | ,, | Kabire | Muhaia | − | − |
| | Muogozi | 25 | M. | ,, | Kagenda | Kagenda | − | − |
| | Muinda | 30 | M. | ,, | Donda | Muquerwa | − | + |
| | Bamutarye | 45 | M. | ,, | Kaima | Dukadyi | − | + |
| | Mitiekula | 25 | M. | ,, | Keango | Kabodyi | − | − |
| ,, 17 | Kida | 25 | M. | Busiro | Luoka | Mukulu | − | + |
| | Zambatise | 25 | M. | ,, | Kiengaza | ,, | − | − |
| | Ibu | 25 | M. | ,, | Luoka | ,, | − | + |
| | Zinazi | 20 | M. | ,, | Kisiba | Kasola | − | − |
| | Takaridia | 20 | M. | Singo | Bujonkola | Kajongolo | − | − |
| | Malugyo | 20 | M. | ,, | Ksagembi | Mbugano | − | − |
| | Bafirawala | 20 | M. | ,, | Kalungu | Mutesa | − | − |
| | Buzibwa | 20 | M. | Busiro | Kireha | Kikwabanga | − | − |
| | Nume | 35 | M. | Singo | Kagembe | Mukwenda | − | + |
| ,, 18 | Kirimentao | 40 | M. | Unyoro | Buianja | Kimbaranga | − | − |
| | Tamanya | 40 | M. | ,, | ,, | ,, | − | − |
| | Makogoto | 40 | M. | ,, | Kikumbia | ,, | − | − |
| | Sabagabu | 25 | M. | ,, | Seresi | Museresi | − | − |
| | Lukala | 25 | M. | ,, | Paehwa | Kimbaranga | − | − |
| | Kowemi | 25 | M. | ,, | Bubangu | Kasoro | − | − |
| | Kahirebu | 35 | M. | ,, | Buiauja | Kimbaranga | − | − |
| | Rukubywa | 40 | M. | ,, | Paehwa | ,, | − | − |
| | Kieka | 20 | M. | ,, | ,, | Peteri | − | − |
| | Murumbia | 30 | M. | ,, | .. | ,, | − | + |
| ,, 19 | Zezefu | 25 | M. | Bulemwezi | Kaswa | Kasiba | − | − |
| | Katagu | 18 | M. | Buddu | Kagandu | Kakembo | − | − |
| | Mukasa | 14 | M. | Busiro | Maganja | Muduauliba | − | − |
| | Name unknown | 20 | M. | (?) | (?) | Bruei | − | − |

### *Incidence of Trypanosomes in the Blood of General Population.*

### C.—Non-Sleeping Sickness Area, Nairobi, British East Africa.

(Examined by Capt. Greig, I.M.S., and Dr. Wiggins.)

| Date. | Name. | Occupation. | District. | Trypanosoma. | Filaria. |
|---|---|---|---|---|---|
| July 14 | Sinandi | Askaris | Masai | — | — |
| | Gila | ,, | ,, | — | — |
| | Massina | ,, | ,, | — | — |
| | Barugo | ,, | ,, | — | — |
| | Matolo | ,, | ,, | — | — |
| ,, 15 | Nandi | ,, | ,, | — | — |
| ,, 15 | Tomana | ,, | ,, | — | — |
| | Rosani | ,, | ,, | — | — |
| | Rosi | ,, | ,, | — | — |
| | Lendiana | ,, | ,, | — | — |
| | Gingili | ,, | ,, | — | — |
| ,, 16 | Rangue | ,, | ,, | — | — |
| | Kabode | ,, | ,, | — | — |
| | Kamia | ,, | ,, | — | — |
| | Tabangi | ,, | ,, | — | — |
| ,, 17 | Karmongi | Labourer | Kukuyu | — | — |
| | Yurimgo | ,, | ,, | — | — |
| | Girewamatwo | ,, | ,, | — | — |
| | Katono | ,, | ,, | — | — |
| | Gomi | ,, | ,, | — | — |
| | Kayaro | ,, | ,, | — | — |
| ,, 18 | Ati | Prisoner | ,, | — | — |
| ,, 20 | Gmosi | ,, | ,, | — | — |
| | Luwi | ,, | ,, | — | — |
| | ? | ,, | Masai | — | — |
| | Nana | ,, | ,, | — | — |
| | Seri | ,, | ,, | — | — |
| | Goma | ,, | ,, | — | — |
| ,, 20 | Kinare | ,, | ,, | — | — |
| | Mahone | ,, | Kukuyu | — | — |
| ,, 21 | Masagai | ,, | Masai | — | — |
| | Desa | ,, | ,, | — | — |
| | Muenda | ,, | ,, | — | — |
| | Morandat | ,, | ,, | — | — |
| | Joke | ,, | Wakamba | — | — |
| | Waweme | ,, | Masai | — | — |
| | Zabe | ,, | Wakamba | — | — |
| | Kalango | ,, | Kukuyu | — | — |
| | Harya | ,, | ,, | — | — |
| ,, 22 | Nabo | Askaris | Masai | — | — |
| | Legina | ,, | ,, | — | — |
| | Kisikas | ,, | ,, | — | — |
| | Mandasingere | ,, | ,, | — | — |
| | Magome | ,, | ,, | — | — |
| | Longotoematabut | ,, | ,, | — | — |
| | Gomi | ,, | ,, | — | — |
| | Lulgini | ,, | ,, | — | — |
| | Zeru | Labourer | Kukuyu | — | — |
| | Jarogo | ,, | ,, | — | — |
| | Kanosi | ,, | ,, | — | — |

Eighty natives from the Sleeping Sickness area were examined, with the result that 23 were found to have trypanosomes in their blood, giving a percentage of 28·7.

One hundred and seventeen were examined from Non-Sleeping Sickness areas, but not a single trypanosoma was found.

We submit that these results suggest that the trypanosomes found in the blood of seemingly healthy persons, and in the cerebro-spinal fluid of Sleeping Sickness cases are identical, and further, that these trypanosomes are in truth the cause of the disease.

### 7. *No Difference can be made out Microscopically between the Trypanosoma of Sleeping Sickness and that of Trypanosoma Fever.*

In the Progress Report it was stated that it would appear that the trypanosomes found in Sleeping Sickness are shorter, have chromatic dots more frequently, and the micro-nucleus is situated nearer the end than in the other.

Since the above was written, an observation has been made, which would seem to do away with any significance the above differences in size and shape may seem to have. It is that trypanosomes from the cerebro-spinal fluid injected into the blood of monkeys become quite as long as the trypanosomes found in the blood of man, and the other differences also disappear. This would go to show that the trypanosomes merely find the cerebro-spinal fluid not as favourable for their growth as the blood, and on this account are stunted.

### 8. *Are these Trypanosomes pathogenic to Animals and can any Specific Difference be made out between them by Animal Experiment?*

The only notice we have seen of the result of the inoculation of animals with the West African *Trypanosoma Gambiense* (Dutton), is that recorded by Annett in the "British Medical Journal," February 7, 1903.

He states "the disease is very chronic in white rats; some appear to be refractory, others died in two or three months. Similarly also in monkeys the infection only proves fatal in about 2 months. Dogs seem to be entirely refractory."

In the following account of the inoculation of various animals with trypanosomes from the cerebro-spinal fluid of Sleeping Sickness cases and from the blood of persons showing no symptoms of Sleeping Sickness we shall not apologise for giving temperature charts and entering into details. In a new and little-known disease descriptions of experiments should be given with as much detail as possible.

The animals available here for inoculation are monkeys, dogs, guinea-pigs, rats, donkeys, oxen, sheep and goats. No animal has been found which shows any marked susceptibility to this disease, or in which the disease runs a rapid acute course, as is the case in Nagana.

The monkeys are the most useful, and they have been injected subcutaneously, into the vertebral canal, and through the foramen magnum into the brain cavity, with cerebro-spinal fluid and blood from many sources.

### A.—*Experiments on the Effect on Monkeys of the Injection of Cerebro-Spinal Fluid containing Trypanosomes taken by Lumbar Puncture from Cases of Sleeping Sickness.*

EXPERIMENT 1.—Monkey, Male, Pale-faced variety (*Macacus rhesus*).

To note the effect of the subcutaneous injection of cerebro-spinal fluid containing trypanosomes, from a case of Sleeping Sickness.

March 23, 1903. Injected 10 c.c. of cerebro-spinal fluid taken *post-mortem* from Case 18, Kaperi, under the skin of the left side of the monkey.

May 11. As the injection of cerebro-spinal fluid on March 23 has not given positive results, this monkey was again injected under the skin with 2 c.c. of cerebro-spinal fluid from Case 41, Warosansa.

May 28. Trypanosomes were found in the blood for the first time. This is evidently due to the second injection 17 days ago.

June 3. This monkey has been ill during the last week and has gradually been growing thinner and weaker. This afternoon he had a convulsive seizure, which affects the right side more than the left, he has clonic spasms of the right arm and leg with twitching of the face and eyes.

June 4. The trypanosomes are numerous in the blood. His condition has considerably improved. There is no sign of paralysis. The right knee jerk is diminished. He is able to climb into his box.

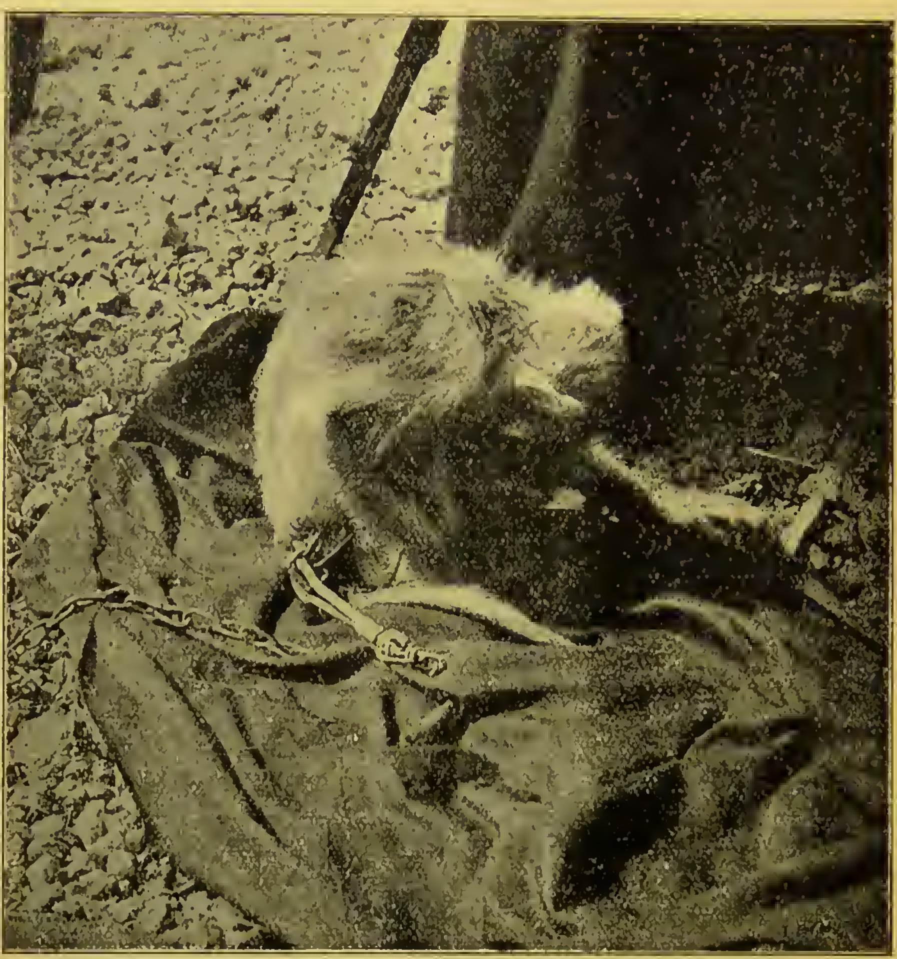
June 5. He is quite lively to-day.

June 11. The trypanosomes are still more numerous.

June 18. The trypanosomes are scanty.

June 23. During the last 10 days this monkey has shown symptoms of Sleeping Sickness. He sits all day long with his head sunk on his chest evidently asleep.

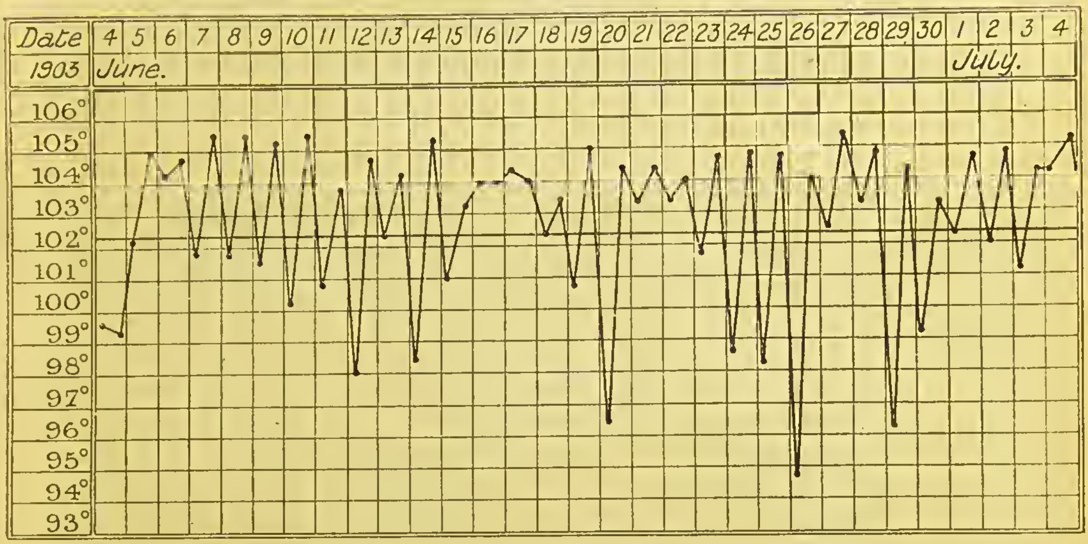
The following photograph shows his appearance at this time:—

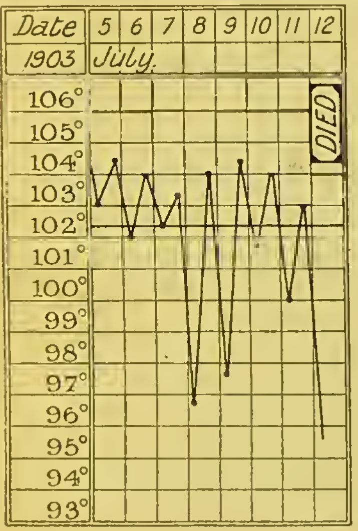


July 10. Since the last entry the appearance of Sleeping Sickness has become more marked. The animal sits still all day with eyes closed and head sunk on chest. He is extremely emaciated but has no marked signs of paresis. He can still climb into his box. As he sits in this lethargic condition the muscles of the arms and trunk frequently show marked twitching, a symptom often seen in the last stages of Sleeping Sickness.

July 12. Died at 11.30 A.M.

The following chart represents the temperature curve from June 4 to July 12. From March 24 to May 23 the temperature remained absolutely normal. No observations were made from May 24 to June 3.





The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| Apr. 7.......... | | – | – |
| ,, 8.......... | | – | – |
| ,, 9.......... | | – | – |
| ,, 11.......... | | – | – |
| ,, 14.......... | | | – |
| ,, 22.......... | – | – | – |
| ,, 30.......... | – | – | – |
| May 7.......... | | | – |
| ,, 14.......... | | – | – |
| ,, 21.......... | | – | – |
| ,, 28.......... | | – | + |
| June 4.......... | | – | + |
| ,, 11.......... | | – | + |
| ,, 18.......... | | – | + |
| ,, 25.......... | | – | + |
| July 1........ | | – | + |
| ,, 10.......... | | – | + |

*Post-mortem* immediately after death.

The body is extremely emaciated. There is an ulcer in the right groin about the size of a shilling. The inguinal and axillary glands are enlarged. The mucous membranes are pale. The right pupil is dilated, the left is normal.

On opening the body the omentum is seen to be studded thickly with hard tubercular nodules, and the surface of the liver and lungs are also seen to be tubercular.

On removing the calvarium, the dura mater is found to be healthy. On the surface of the left cerebral hemisphere there is seen a caseous mass about the size of a bean which is adherent to the dura mater and evidently tuberculous in origin. The surface of the brain is pale in colour and the vessels are only slightly injected. Unfortunately, owing to an accident, the brain was not preserved for more minute examination.

Microscopical examination of blood from the heart showed numerous trypanosomes.

*Remarks.*—During life this animal presented the appearance of a typical Sleeping Sickness case. Even when in the act of eating, his eyes would close, his head fall forward on his chest, and he would sit apparently sound asleep. It was disappointing to find on *post-mortem* examination that the animal was tubercular.

It is to be regretted that the brain was not preserved, also that no examination of cerebro-spinal fluid was made for trypanosomes. Nevertheless, when this case is placed beside Experiment 60 (p. 55),* and the two are compared, it will be agreed that this monkey owed its symptoms to the trypanosomes and not to the tubercle.

This pale-faced variety was brought by us from England and none of them harbour malarial parasites as most of the local black-faced variety do. They seem to be more susceptible to the trypanosomes than the local species.

### EXPERIMENT 34.—Monkey (Male) (*Macacus rhesus*).

To note the effect of injecting the cerebro-spinal fluid from a case of Sleeping Sickness into the vertebral canal of a monkey.

April 8, 1903. Injected 1 c.c. of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into the spinal canal of this monkey.

April 30. Trypanosomes appeared in the blood to-day, 19 days after inoculation into the spinal canal. Note that the temperature curve shows no sign of this invasion.

May 21. Trypanosomes numerous in the blood. Temperature taken and found to be 106°·4 F.

Aug. 25. This monkey is beginning to show the usual symptoms of the disease in the monkey. He sits most of the day with his head fallen on his chest, evidently asleep, and his temperature has become very irregular.

The following chart represents the temperature curve :—

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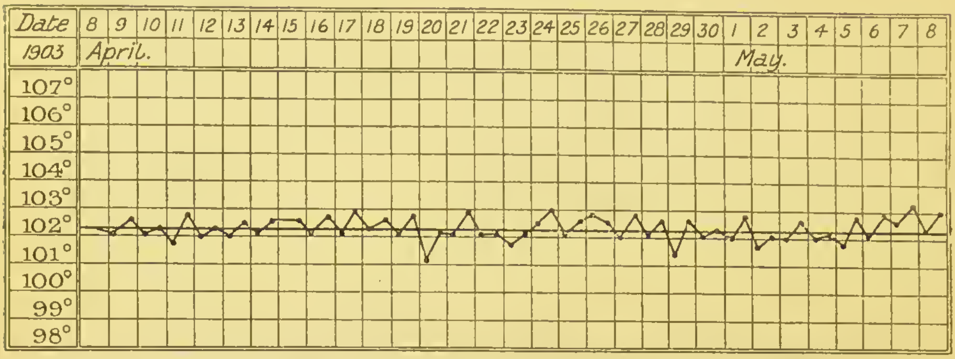
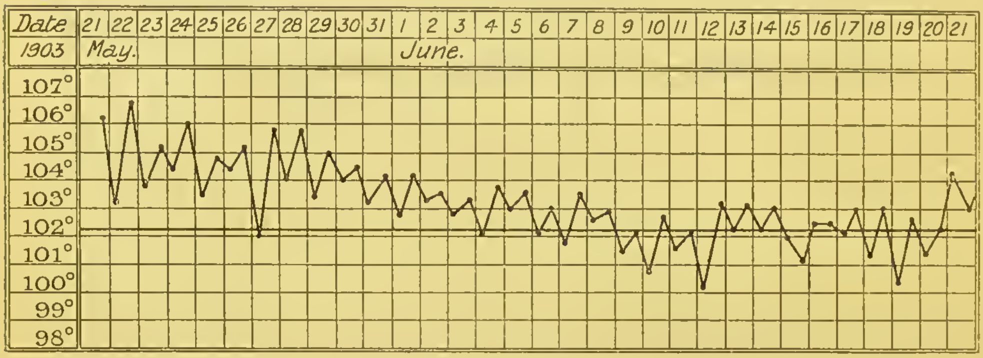
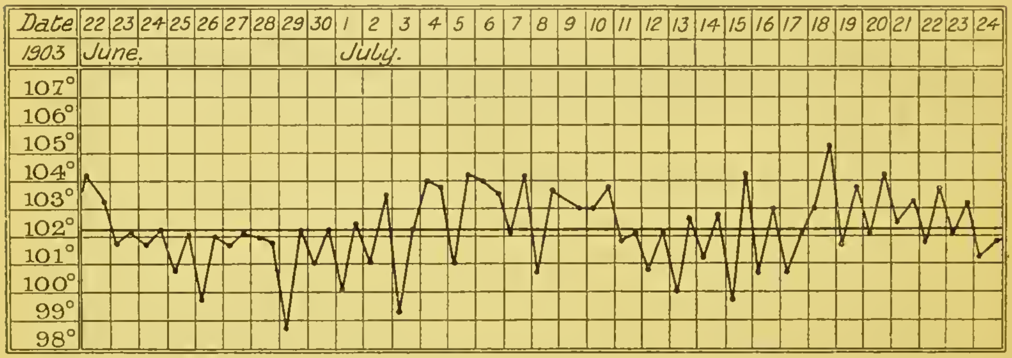
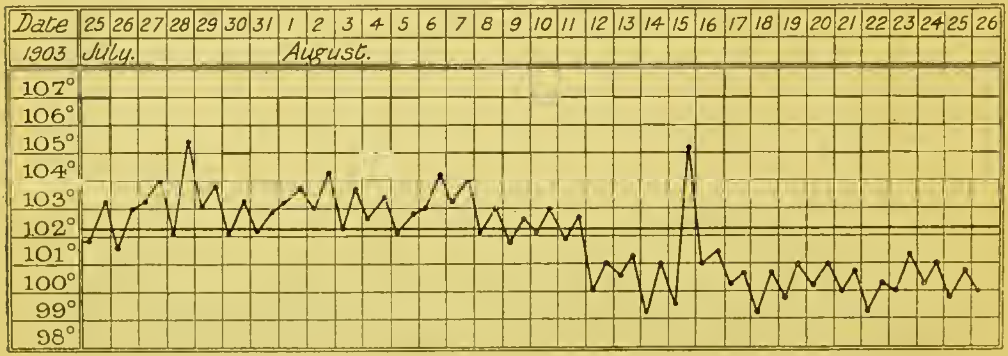
* See Progress Report.


Date
1903
8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8
April.
May.
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°


Date
1903
21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
May.
June.
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°


Date
1903
22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
June.
July.
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°


Date
1903
25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26
July.
August.
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°

The following table shows the presence or absence of trypanosomes in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| Apr. 11.......... | — | — | — |
| ,, 24.......... | — | — | — |
| ,, 30.......... | — | — | + |
| May 7.......... | | — | + |
| ,, 14.......... | | — | + |
| ,, 21 ........ | | — | + |
| ,; 27.......... | | — | + |
| June 4 ......... | | — | + |
| ,, 11.......... | | — | + |
| ,, 18.......... | | — | + |
| ,, 25.......... | | — | + |
| July 1.......... | | — | + |
| ,, 23.......... | | — | + |
| ,, 31.......... | | — | + |
| Aug. 13.......... | | — | + |
| ,, 20.......... | | — | + |

*Remarks.*—Four months after inoculation this monkey begins to show symptoms of Sleeping Sickness. It is to be regretted that it was considered impossible to draw off and examine the cerebro-spinal fluid of the monkey on account of the small size of these animals. With care and practice probably it could be done.*

EXPERIMENT 95.—Monkey, black-faced variety (*Cercopithecus* sp.).

To note the effect of injecting the cerebro-spinal fluid from a case of Sleeping Sickness into the vertebral canal of a monkey.

May 13, 1903. Blood examined; there were no malarial parasites or trypanosomes present.

May 14. Injected 1 c.c. of cerebro-spinal fluid from a case of Sleeping Sickness into the vertebral canal of this monkey.

The following table shows the presence or absence of trypanosomes in the blood:—

---

* Captain Greig writes on September 10 that No. 34 has died. "*Post-mortem* was pretty typical of an ordinary Sleeping Sickness case. Trypanosomes were found living in cerebro-spinal fluid of brain."

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 22.......... | | – | – |
| ,, 29.......... | | – | – |
| June 4*........ | | – | + |
| ,, 11.......... | | – | – |
| ,, 20.......... | | – | + |
| ,, 25.......... | | – | + |
| July 1.......... | | – | + |
| ,, 23.......... | | – | – |
| ,, 31.......... | | – | + |
| Aug. 13.......... | | – | + |
| ,, 21.......... | | – | + |

*Remarks.*—Up to the end of August this animal has shown no symptoms of Sleeping Sickness.

### EXPERIMENT 96.—Monkey (*Cercopithecus* sp.).

To note the effect of injecting the cerebro-spinal fluid from a case of Sleeping Sickness into the vertebral canal of a monkey

May 14, 1903. Blood examined. No malaria or trypanosoma.

Injected 1 c.c. of cerebro-spinal fluid from a case of Sleeping Sickness into the vertebral canal of this monkey.

August 25. No symptoms. No rise of temperature.

The following table shows the presence or absence of trypanosomes in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 22.......... | | + | – |
| ,, 29.......... | | + | – |
| June 4.......... | | + | – |
| ,, 11.......... | | + | – |
| ,, 20.......... | | + | – |

June 24. Again injected 1 c.c. of cerebro-spinal fluid from a case of Sleeping Sickness into the spinal canal of this monkey.

* Twenty-one days after inoculation.

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| July 1 | | + | − |
| ,, 23 | | + | − |
| ,, 31 | | + | + |
| Aug. 13 | | + | + |
| ,, 21 | | + | + |

EXPERIMENT 54.—Monkey (*Cercopithecus* sp.).

To note effect of injection of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into brain cavity of a monkey.

April 9, 1903. Blood examined. No trypanosoma or malaria.

Injected 1 c.c. of cerebro-spinal fluid from a case of Sleeping Sickness into the brain of this monkey through the foramen magnum.

August 25. No symptoms up to present date. The temperature has remained normal throughout.

The following table shows the presence or absence of trypanosomes in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| Apr. 11 | | − | − |
| ,, 23 | − | − | − |
| ,, 30 | − | − | + |
| May 7 | | − | + |
| ,, 14 | | − | + |
| ,, 21 | | − | + |
| ,, 28 | | − | + |
| June 4 | | − | + |
| ,, 11 | | − | + |
| ,, 18 | | − | + |
| ,, 25 | | + | + |
| July 1 | | − | + |
| ,, 23 | | − | + |
| ,, 31 | | − | + |
| Aug. 13 | | − | − |
| ,, 20 | | − | + |

## B.—*Experiments on the Effect of the Injection into Monkeys of Blood containing Trypanosomes from Cases showing no Symptoms of Sleeping Sickness.*

### Experiment 60.—Monkey (*Macacus rhesus*).

To note the effect of the subcutaneous injection of blood from a case of trypanosoma fever.

April 15. Injected subcutaneously 2 c.c. of blood containing trypanosomes from Case 66, Tabula, Marine.

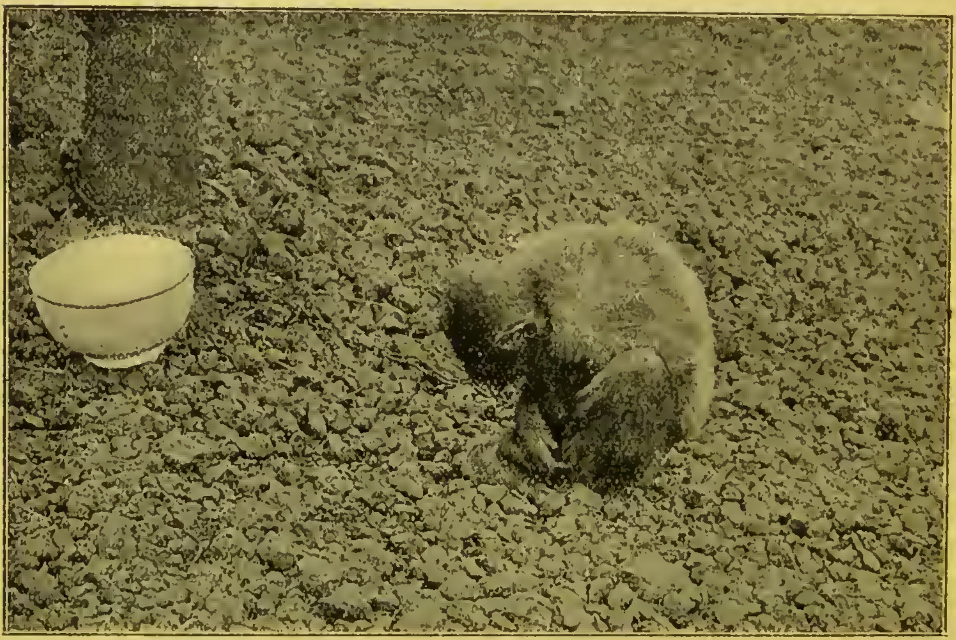
May 7. Trypanosomes appeared in the blood for the first time, 22 days after injection.

May 14. The hæmatozoa are noted as being very numerous.

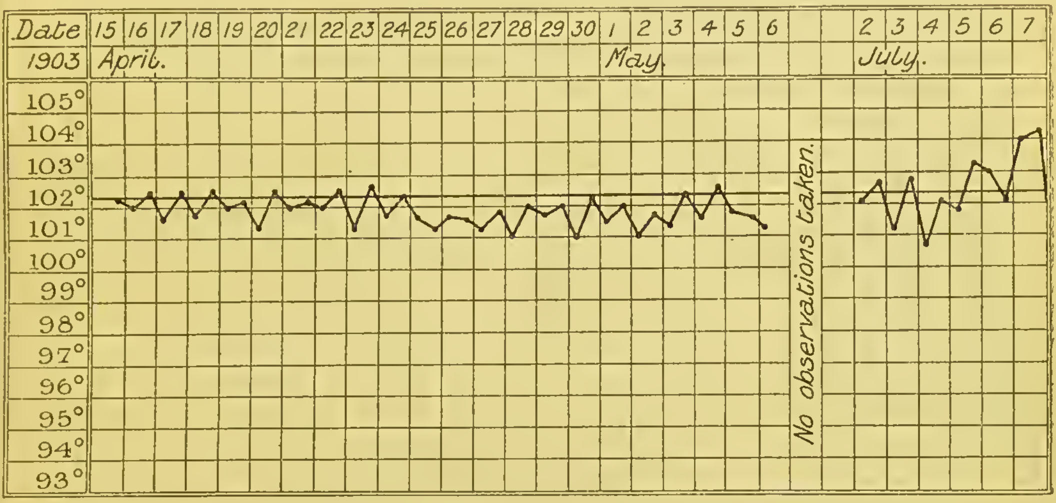
July 2. Up to the present this monkey has shown no signs of being ill. To-day, however, he appears listless and less energetic. Temperature observations resumed.

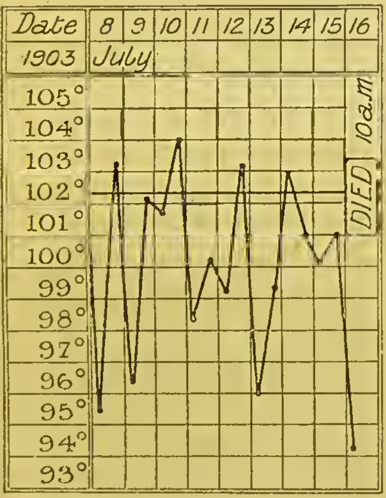
July 15. For the last fortnight this monkey has presented the same picture of Sleeping Sickness as noted in the case of Experiment 1, monkey. He sits about all day with his head sunk on his chest, evidently asleep. He has become emaciated and to some extent puffy and cyanosed about the face.

The following photograph shows his appearance at this time :—



The following chart represents the temperature curve. From April 15 to May 6 the temperature remained normal. No observations were made from May 6 to July 2, when observations were resumed.





The following table shows the presence or absence of trypanosomes in the blood and cerebro-spinal fluid :—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 15 .... | | — | | | |
| „ 23 .... | — | — | — | | |
| „ 30 .... | — | — | — | | |
| May 7 .... | | — | + | | |
| „ 14 .... | | — | + | | |
| „ 21 .... | | — | + | | |
| „ 28 .... | | — | + | | |
| June 4 .... | | — | + | | |
| „ 11 .... | | — | + | | |
| „ 20 .... | | — | + | | |
| „ 25 .... | | — | + | | |
| July 1 .... | | — | + | | |
| „ 16 .... | | — | + | | + |

July 16. Died at 10 A.M. *Post-mortem* immediately after death.

The body is much emaciated. The pupils are equal and normal. The axillary, inguinal and lymphatic glands are enlarged.

On opening the body there is no fluid found in the peritoneal, pericardial or pleural cavities. There are no signs of tubercle in the mesentery, spleen, liver or lungs.

On removing the calvarium, the dura mater is found to be normal. On reflecting this membrane the surface of the brain presents a striking resemblance to that found in man in cases of Sleeping Sickness. The convolutions are flattened; there is some injection of the superficial vessels; the sub-arachnoid fluid is in excess, filling up the sulci and giving them the ground-glass appearance so often described in cases of Sleeping Sickness. Fluid taken from the lateral ventricle and examined microscopically shows trypanosomes. The brain was removed entire and preserved for future minute examination.

The *heart* is normal in appearance. Blood taken from it and examined microscopically shows many living trypanosomes, many of which present peculiar forms rarely met with. Several of these are represented in the coloured plate.

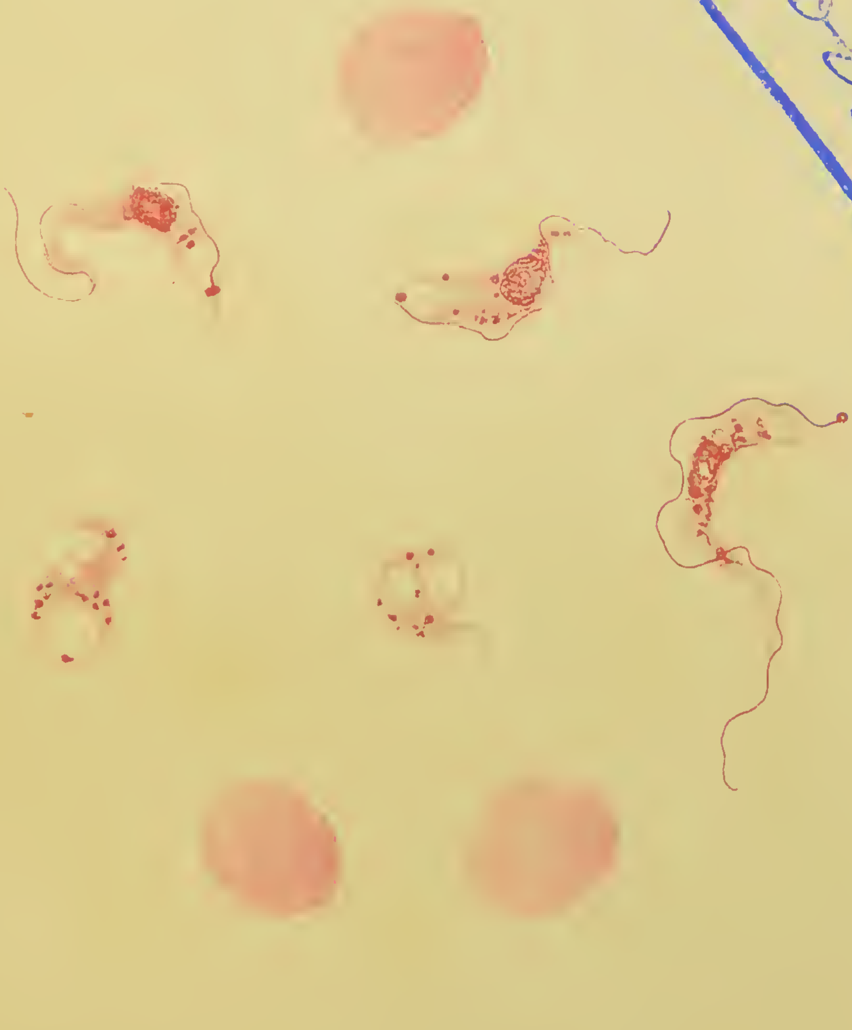
The *lungs* show no signs of tuberculosis. They are somewhat congested, otherwise normal.

The *liver* is congested and presents a nutmeg appearance.

The *spleen* is large, dark in colour and firm in consistence.

The *kidneys* are normal. The *intestines* appear healthy and present nothing noteworthy macroscopically.

*Bruce & Nabarro.* *Sleeping Sickness Further Report, Pl.1*



[illegible] lith et imp

*Remarks.*—This is a noteworthy case and should be compared with monkey, Experiment 1 (p. 36).* As far as one could judge, this animal presented the typical appearance of Sleeping Sickness during life, and the brain after death looked like the usual Sleeping Sickness brain in miniature. The organs showed no disease, so that one is bound to look upon the injected trvpanosomes as the cause of death.

But the trypanosomes which were injected subcutaneously into this monkey were taken from the blood of a case of trypanosoma fever, from Case 66, Tabula, who, up to the present (August), has shown no signs of Sleeping Sickness. The trypanosomes, although injected subcutaneously, have found their way into the cerebro-spinal fluid and given rise to symptoms which closely resemble Sleeping Sickness in man.

This result is certainly suggestive, and, in our opinion, goes far to prove that we are dealing with one species of trypanosoma, and that Tabula and the others are in the incipient stage of Sleeping Sickness.

### EXPERIMENT 56. Monkey (*Cercopithecus* sp.).

To note effect of the injection into the vertebral canal of blood containing trypanosomes from Case 66, Tabula, Marine.

April 14, 1903. Examined the blood of this monkey to-day, and found malarial parasites but no trypanosomes.

April 28. Injected by lumbar puncture 2 c.c. of blood from Case 66, Tabula, Marine, containing trypanosomes, into spinal canal.

May 7. Trypanosomes found in the peripheral blood.

No temperature chart was kept in this case.

The following table shows the presence or absence of trypanosoma in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 14 .... | | + | – | | |
| May 7 .... | | + | + | | |
| „ 14 .... | | + | – | | |
| „ 21 .... | | + | – | | |
| „ 30 .... | | + | + | | |
| June 4 .... | | + | + | | |
| „ 11 .... | | + | + | | |
| „ 20 .... | | + | + | | |
| „ 25 .... | | + | + | | |
| July 1 .... | | + | + | | |
| „ 10 .... | | | | | + |

---

* See Progress Report.

July 1. This monkey is slowly becoming more and more emaciated. He eats his food well and has no very marked symptoms of Sleeping Sickness.

July 10. Died at 1 P.M. *Post-mortem* immediately after death.

The body is emaciated. No external sores. Pupils equal and normal. No signs of tuberculosis. The axillary, inguinal, and femoral glands are slightly enlarged. The heart, lungs, liver, spleen, kidneys, and intestines appear fairly normal to the naked eye.

On making a microscopical examination of the cerebro-spinal fluid from the lateral ventricle, living trypanosomes are found in some numbers. The brain presented nothing abnormal to the naked eye and was preserved for minute examination.

*Remarks.*—Nothing can be positively asserted about this case. It is true that the *post-mortem* appearances showed no other cause of death, but during life there were certainly no marked symptoms of Sleeping Sickness, as in Experiments 1 and 60, though it was noted that during the last few weeks of life, he became disinclined to move. Instead of coming to the end of his chain to receive his food, he sat still and waited till it was put into his hand. It may be noted that the trypanosomes appeared in the blood nine days after injection of blood into the spinal canal, and also that living trypanosomes were found in the lateral ventricles after death. Death occurred 71 days after the inoculation, and in our opinion is probably due to the trypanosomes. It is to be regretted that no temperature chart was kept in this case.

### EXPERIMENT 61. Monkey (*Macacus rhesus*).

To note the effect of the injection into the vertebral canal of blood containing trypanosomes from Case 66, Tabula, Marine.

April 28, 1903. Blood examined. No trypanosomes or malaria.

Injected 2 c.c. of blood from Case 66, Tabula, Marine, containing trypanosomes, into the spinal canal of this monkey.

August 25. No symptoms.

The following table shows the presence or absence of trypanosoma in the blood :—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| Apr. 15.......... | | — | — |
| May 7.......... | | — | + |
| ,, 14.......... | | — | + |
| ,, 21.......... | | — | + |
| ,, 28.......... | | — | + |
| June 4.......... | | — | + |
| ,, 11.......... | | — | + |
| ,, 18.......... | | — | + |
| ,, 25.......... | | — | — |
| July 1.......... | | — | + |
| ,, 23.......... | | — | — |
| ,, 31.......... | | — | — |
| Aug. 13.......... | | — | — |
| ,, 20.......... | | — | — |

*Remarks.*—It is curious that no trypanosomes have been seen in the blood during the last month.

### EXPERIMENT 121. Monkey (*Cercopithecus* sp.).

To note effect of injection of blood containing trypanosomes from Case 66, Tabula, into brain of a monkey through foramen magnum.

June 20, 1903. Blood examined. No trypanosoma. Malaria present.

Injected 1 c.c. of blood from Tabula, Marine, into the brain of this monkey.

The following table shows the presence or absence of trypanosoma and malaria in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 30.......... | | + | — |
| June 4.......... | | + | — |
| „ 11.......... | | + | + |
| „ 20.......... | | + | + |
| „ 25......... | | + | + |
| July 1.......... | | + | + |
| „ 23.......... | | + | + |
| „ 31.......... | | + | + |
| Aug. 13.......... | | + | + |
| „ 20.......... | | + | + |

## Experiment 123. Monkey (*Cercopithecus* sp.).

To note effect of injection of blood containing trypanosomes from Case 64, Jordien Murjan, into brain of monkey through foramen magnum.

May 25, 1903. Examined blood. No trypanosoma present.

Injected 1 c.c. of blood from Case 64, Jordien Murjan, into the brain of this monkey.

The following table shows the presence or absence of trypanosoma and malaria in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 28.......... | | + | — |
| June 4.......... | | + | — |
| „ 11.......... | | + | + |
| „ 20.......... | | + | + |
| July 1.......... | | + | + |
| „ 23.......... | | + | + |
| „ 31.......... | | + | + |
| Aug. 13.......... | | — | + |
| „ 21.......... | | + | + |

These experiments show that the trypanosomes derived from the cerebro-spinal fluid of cases of Sleeping Sickness, and the trypanosomes derived from the blood of persons showing no symptoms of Sleeping Sickness can both give rise to a chronic and fatal disease in the monkey which bears a striking resemblance in its last stages to Sleeping Sickness in man.

These experiments therefore also tend to strengthen the evidence that the trypanosomes derived from these two sources are identical, and that trypanosoma fever and Sleeping Sickness are stages of the same disease.

It may be noted here that several of the monkeys whose cases were given in the Progress Report are still under observation. The trypanosomes are still found regularly in their blood, but nothing further noteworthy has happened, so that they are not alluded to in this report.

## *Experiments on the Effect of the Injection of these Trypanosomes into Dogs.*

The native dog in Uganda is not very suitable as an experimental animal. Many of them die of ankylostomiasis before the experiment is finished, and this disease is so common among them that it is difficult to obtain one free from it. The conclusion arrived at from a study of the few cases under observation is that the trypanosomes of Sleeping Sickness and trypanosoma fever can live and multiply for some time in the blood of the dog, but tend to disappear sooner or later, and that they show no difference in their action on the dog.

Further, it would seem that the pup is less susceptible than the adult, just as the calf is less susceptible to piroplasma than grown-up cattle.

### A. *The Injection of Cerebro-Spinal Fluid from Cases of Sleeping Sickness into Dogs.*

This disappearance of the trypanosomes from the blood of the dog is interesting, and makes it a connecting link between the susceptible monkey and the refractory goat, etc.

It is quite possible that there is a certain percentage of men absolutely refractory to the disease, another percentage in which the parasite does not get beyond the blood and is there killed off (*vide* Tabula, Monkey 61) leaving the man immune, and lastly a percentage in which the trypanosomes gain access to the cerebro-spinal fluid, and the symptoms of Sleeping Sickness and a fatal termination supervene.

### EXPERIMENT 85.—Dog. Black and White.

To note effect of subcutaneous injection of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into a dog.

May 5, 1903. Injected 10 c.c. of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into this dog.

August 18. Dog died. Ankylostomiasis.

The following table shows the presence or absence of trypanosoma in the blood :—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | *Filaria immitis.* | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 12 | | | – |
| ,, 19 | | | – |
| ,, 26 | | | – |
| June 2 | + | | – |
| ,, 9 | | | + |
| ,, 16 | | | – |
| ,, 24 | | | – |
| ,, 30 | | | – |
| July 17 | | | – |
| ,, 21 | | | – |
| ,, 28 | | | – |
| Aug. 5 | | | – |
| ,, 14 | | | – |

### EXPERIMENT 49.—Brindled Dog.

April 6, 1903. Injected 1 c.c. of cerebro-spinal fluid, containing trypanosomes from a case of Sleeping Sickness into this dog.

June 16. Died June 17. *Post-mortem.*—No ankylostomiasis. Tumours in stomach wall of spiroptera sanguinolenta. Heart, lungs, liver, and kidneys look healthy. Spleen enlarged.

*Remarks.*—It is impossible to say what was the cause of this dog's death. It may have been the trypanosomes, but this seems most improbable. The temperature curve remained normal throughout, and showed no reaction to the presence of the trypanosomes in the blood.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| Apr. 24 | | | – |
| May 4 | | | + |
| „ 11 | | | – |
| „ 19 | | | + |
| „ 26 | | | + |
| June 2 | | | + (scanty) |
| „ 9 | | | + |
| „ 16 | | | + |

## EXPERIMENT 144.—Brindled Pup.

To note effect of subcutaneous injection of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into a pup.

June 23, 1903. Injected 4 c.c. of cerebro-spinal fluid containing trypanosomes into this pup.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 30 | | | – |
| July 17 | | | – |
| „ 21 | | | – |
| „ 28 | | | – |
| Aug. 4 | | | – |
| „ 14 | | | – |
| „ 18 | | | – |

### Experiment 145.—Fawn-coloured Pup.

June 23, 1903. Injected subcutaneously, 4 c.c. of cerebro-spinal fluid containing trypanosomes into this pup.

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 30.......... | | | – |
| July 17.......... | | | – |

July 19. Died. No trypanosomes in the blood. Died of ankylostomiasis.

## B.—*The Injection of Blood Containing Trypanosomes from Cases Showing no Symptoms of Sleeping Sickness.*

### Experiment 65.—Dog (Brown).

April 15, 1903. Injected subcutaneously 2 c.c. of blood containing trypanosomes from Case 66, Tabula, Marine.

May 11. Dog very emaciated and can hardly stand. Refuses food and is apparently dying. Very anæmic. Paresis of hind extremities. No œdema. No opacity of cornea.

Blood examination.—Red blood corpuscles, 1,700,000 per c.cm.
,, ,, White blood corpuscles, 60,940 ,,
,, ,, Trypanosomes *present* in very small numbers.

Lymphocytes, 34 per cent.; large mononucleated, 12 per cent.; polynucleated, 54 per cent.

Eosinophile cells, 0 per cent.; nucleated red blood corpuscles, 6 per cent.

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| Apr. 24.......... | | | – |
| ,, 27.......... | | | – |
| May 4.......... | | | + |
| ,, 11.......... | | | + |

May 12th. Died 10.15 A.M. Autopsy immediately after death.

The blood is extremely pale and watery. All the organs are bloodless but healthy. The œsophagus shows a small tumour about 2 inches above the cardiac orifice of the stomach, which contains spiroptera sanguinolenta. The wall of the stomach also shows from 15 to 20 tumours of the same parasite, some of them as large as a walnut. The intestine is the seat of innumerable uncinaria trigonocephala, and presents a remarkable appearance. Four inches from the pylorus these ankylostomata begin, and they become more and more numerous until in the ilium they exist in thousands. The greater part of the small intestine is coloured a deep dusky red by numerous hæmorrhages, and the large intestine is full of black blood of the consistency and appearance of tar.

*Remarks.*—The temperature curve remained fairly normal until within a few days of death, when it became sub-normal. Death was due to ankylostomiasis.

EXPERIMENT 72.—Dog. Small Black and Tan. Male.

April 28. Injected 2 c.c. of blood containing trypanosomes from Case 66, Tabula, Marine.

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 11.......... | | | + |
| ,, 19.......... | | | + |
| ,, 26.......... | | | + |
| June 2.......... | | | + |

June 7. Died from anæmia, due to ankylostomiasis.

EXPERIMENT 122.—Dog. Small Black and Tan.

May 20, 1903. Injected subcutaneously 10 c.c. of blood from Case 66, Tabula, Marine.

The following table shows the presence or absence of trypanosoma in the blood :—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 2.......... | | | + |
| „ 9.......... | | | + |
| „ 16.......... | | | — |
| „ 24.......... | | | — |
| „ 30.......... | | | — |
| July 17.......... | | | — |
| „ 21.......... | | | — |
| „ 28.......... | | | — |
| Aug. 4.......... | | | — |
| „ 14.......... | | | — |
| „ 18.......... | | | — |

### EXPERIMENT 146.—Black Pup.

June 23, 1903. Injected subcutaneously 4 c.c. of blood from Case 64, Jordien Murjan, into this dog.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 30.......... | | | — |
| July 17.......... | | | — |
| „ 21.......... | | | — |
| „ 28.......... | | | — |
| Aug. 4.......... | | | — |
| „ 14.......... | | | — |
| „ 18.......... | | | — |

### EXPERIMENT 147.—Black and White Pup.

June 23, 1903. Injected subcutaneously 4 c.c. of blood from Case 64, Jordien Murjan, into this dog.

July 16. Dog died.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 23.......... | | | — |
| „ 30.......... | | | — |
| July 16.......... | | | — |

*On the Effect of the Injection of these Trypanosomes into Guinea Pigs, Donkeys, Oxen, Sheep and Goats.*

### Experiment 82.—Guinea Pig.

To note effect of subcutaneous injection of cerebro-spinal fluid containing trypanosomes from case of Sleeping Sickness into guinea pig.

May 5, 1903. Injected 5 c.c. of cerebro-spinal fluid containing trypanosomes into this guinea pig subcutaneously.

The following table shows the presence or absence of trypanosomes in the blood. Temperature curve remained normal throughout:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 12.......... | | | — |
| „ 19.......... | | | — |
| „ 26.......... | | | — |
| June 2.......... | | | — |
| „ 9.......... | | | — |
| „ 16.......... | | | — |
| „ 24.......... | | | — |
| „ 30.......... | | | — |
| July 17.......... | | | — |
| „ 21.......... | | | — |
| „ 28.......... | | | — |
| Aug. 4.......... | | | — |
| „ 14.......... | | | — |
| „ 18.......... | | | — |

### Experiment 81.—Guinea Pig (Female).

To note effect of subcutaneous injection of blood containing trypanosomes from man showing no signs of Sleeping Sickness into guinea pig.

May 1, 1903. Injected $\frac{1}{2}$ c.c. of blood from Case 66, Jordien Murjan, containing trypanosomes.

The following table shows the presence or absence of trypanosomes in the blood. Temperature curve remained normal throughout:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 11 | | | — |
| ,, 19 | | — | — |
| ,, 26 | | | — |
| June 2 | | | — |
| ,, 9 | | | — |
| ,, 16 | | | — |
| ,, 24 | | | — |
| ,, 30 | | | — |
| July 21 | | | — |
| Aug. 4 | | | — |
| ,, 14 | | | — |
| ,, 18 | | | — |

### Experiment 100.—Donkey.

To note effect of subcutaneous injection of cerebro-spinal fluid containing trypanosomes from case of Sleeping Sickness into a donkey.

May 14, 1903. Injected subcutaneously 6 c.c. of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into this donkey.

The following table shows the presence or absence of trypanosomes in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 14.......... | | | — |
| ,, 23.......... | | | — |
| ,, 27.......... | | | — |
| June 3 ......... | | | — |
| ,, 10.......... | | | — |
| ,, 17.......... | | | — |
| ,, 24 ......... | | | — |
| July 1.......... | | | — |
| ,, 17.......... | | | — |
| ,, 22.......... | | | — |
| Aug. 5.......... | | | — |
| ,, 13.......... | | | — |
| ,, 19.......... | | | — |

### EXPERIMENT 101.—Donkey.

To note effect of subcutaneous injection of blood containing trypanosomes from man showing no signs of Sleeping Sickness into donkey.

May 11, 1903. Examined blood. No trypanosomes. No malaria.

May 15. Injected 10 c.c. of blood subcutaneously from Case 68, Bara Risgallah, into this donkey.

May 26. Again injected 10 c.c. of blood from Case 68.

May 27. To-day a large swelling was noticed in the region of the second inoculation.

June 5. Abscess opened, several ounces of pus evacuated.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 23.......... | | | — |
| ,, 27.......... | | | — |
| June 3.......... | | | — |
| ,, 10.......... | | | — |
| ,, 17.......... | | | — |
| ,, 24.......... | | | — |
| July 1.......... | | | — |
| ,, 17.......... | | | — |
| ,, 22.......... | | | — |
| Aug. 5.......... | | | — |
| ,, 13.......... | | | — |
| ,, 19.......... | | | — |

### EXPERIMENT 112.—Ox. Small yellow.

To note effect of subcutaneous injection of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into oxen.

May 18, 1903. Blood examined. No trypanosoma. No other parasites.

Injected 10 c.c. of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into this ox.

June 14. 10 c.c. of blood was taken and centrifuged; it was pale, and laked readily. No trypanosomes were found.

June 15. Died this morning.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 27.......... | | | — |
| June 3.......... | | | — |
| ,, 10.......... | | | — |
| ,, 14.......... | | | — |

*Post-mortem.*—Lymphatic glands much enlarged.

*Heart.*—At base a collection of yellow jelly-like material. This jelly-like material is found subcutaneously, and in the pelves of the kidneys, etc. This disease is known locally as (M'Kebbe), and an owner of cattle here informs us that 90 per cent. of deaths among his cattle are due to this.

This is an Ankole ox, and, therefore, said to be more liable to this disease. Cattle reared in this district are looked upon as less susceptible. Is this African Coast fever?

### EXPERIMENT 132.—Ox. Small yellow.

June 17, 1903. Blood examined. Trypanosomes absent.

June 21. Injected subcutaneously 10 c.c. of cerebro-spinal fluid, containing trypanosomes from a case of Sleeping Sickness, into this ox.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 24.......... | | | – |
| July 1.......... | | | – |
| „ 17.......... | | | – |
| „ 22.......... | | | – |
| Aug. 5.......... | | | – |
| „ 13.......... | | | – |
| „ 19.......... | | | – |

EXPERIMENT 111.—Ox. Big brown.

To note effect of subcutaneous injection of blood, containing trypanosomes from a man showing no signs of Sleeping Sickness, into an ox.

May 18, 1903. Examined the blood. Trypanosoma absent. No parasites seen.

May 19. Injected 10 c.c. of blood from Case 31, Karala Barigi, containing trypanosomes, into this ox.

May 25. Injected 5 c.c. of blood from Case 64, Jordien Murjan.

June 21. Died this morning.

*Post-mortem.* The same appearance as Experiment 112 (M'Kebbe).

The following table shows the presence or absence of trypanosoma in the blood :—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 27.......... | | | – |
| June 3.......... | | | – |
| „ 10.......... | | | – |
| „ 17.......... | | | – |

That oxen in Entebbe do harbour trypanosomes is proved by the fact that, of three sent to us by Mr. Pordage, P.W.D., for examination, two were found to have trypanosomes in their blood. These cattle were emaciated, their hair stared, and they looked decidedly out of health. As the trypanosomes in the blood of these oxen might be the Nagana parasite, 2 c.c. of blood from one of them was injected into a dog. The following experiment shows with what result :—

### Experiment 128.—Dog. Brown.

To note effect of subcutaneous injection of blood from Mr. Pordage's brown ox, which was found to contain trypanosomes, into a dog.

June 3, 1903. Injected subcutaneously 2 c.c. of blood.

August 16. Re-injected 5 c.c. blood from same ox, containing trypanosomes.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| June 9.......... | | | — |
| ,, 16.......... | | | — |
| ,, 24.......... | | | — |
| ,, 30.......... | | | — |
| July 17.......... | | | — |
| ,, 21.......... | | | — |
| ,, 28.......... | | | — |
| Aug. 4.......... | | | — |
| ,, 14.......... | | | — |
| ,, 18.......... | | | — |
| | | | — |

Mr. Pordage informs us that these oxen came to Entebbe from British East Africa about the end of 1900. They kept remarkably well for more than two years. During this time they were grazed on high ground away from the lake shore. About the beginning of 1903 they were sent to graze in and about the forest, which fringes the lake. To this circumstance Mr. Pordage attributes their illness. It is impossible at present to say what species of trypanosoma these cattle harbour. It does not seem to be Nagana, or the dog would have died. On the other hand, we have failed to inoculate *young* oxen with the trypanosomes of Sleeping Sickness.

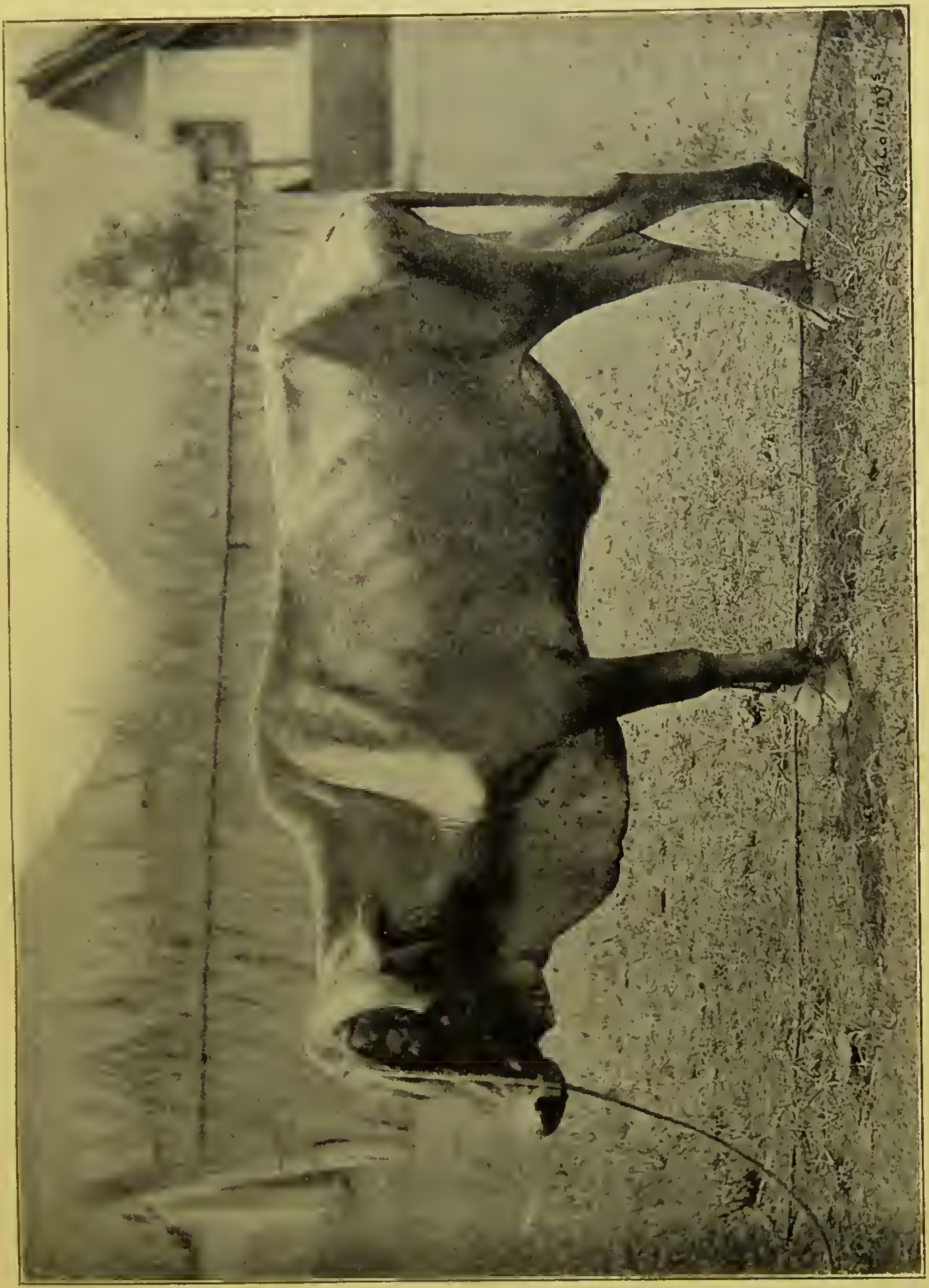
Further experiments are being made to try to decide this question.

The accompanying plate shows the appearance of Mr. Pordage's ox.

### Experiment 150.—Goat.

To note effect of subcutaneous injection of cerebro-spinal fluid, containing trypanosomes from a case of Sleeping Sickness into goats.

June 30, 1903. Injected 10 c.c. of cerebro-spinal fluid, from a case of Sleeping Sickness, into this goat.



Mr. Pordage's Ox.

July 1. Trypanosomes absent.
July 17. Trypanosomes absent.
July 22. Goat died.

*Post-mortem.*—Much fluid in the pericardium. Lungs have the same appearance as in South African horse sickness. Lymphatic glands much enlarged generally. Yellow jelly-like material at base of the heart.

### EXPERIMENT 90.—Goat.

To note effect of subcutaneous injection of blood, containing trypanosomes from a man showing no signs of Sleeping Sickness into a goat.

May 11, 1903. Injected subcutaneously 5 c.c. of blood from Case 64, Jordien Murjan, containing trypanosomes, into this goat.

The following table shows the presence or absence of trypanosoma in the blood.

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 20.......... | | — | — |
| ,, 27.......... | | | — |
| June 3.......... | | | — |
| ,, 10.......... | | | — |
| ,, 17.......... | | | — |
| ,, 24.......... | | | — |
| July 1.......... | | | — |
| ,, 17.......... | | | — |
| ,, 22.......... | | | — |
| Aug. 1.......... | | | — |
| ,, 13.......... | | | — |
| ,, 19.......... | | | — |

### EXPERIMENT 89.—Sheep. Brown.

To note effect of subcutaneous injection of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into a sheep.

May 11, 1903. Injected 10 c.c. of cerebro-spinal fluid containing trypanosomes from a case of Sleeping Sickness into this sheep.

June 30. Again injected 10 c.c. of fluid.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| May 20 | | | — |
| ,, 27 | | | — |
| June 3 | | | — |
| ,, 10 | | | — |
| ,, 17 | | | — |
| ,, 24 | | | — |
| July 1 | | | — |
| ,, 17 | | | — |
| ,, 22 | | | — |
| Aug. 5 | | | — |
| ,, 13 | | | — |
| ,, 19 | | | — |

EXPERIMENT 149.—Sheep. Small Brown and White.

To note effect of subcutaneous injection of blood containing trypanosomes from a man showing no signs of Sleeping Sickness into sheep.

June 25, 1903. Injected subcutaneously 5 c.c. of blood from Case 63, Kumsarsabba, into this sheep.

The following table shows the presence or absence of trypanosoma in the blood:—

| Date. | Parasites in blood. | | |
|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. |
| 1903. | | | |
| July 1 | | | — |
| ,, 17 | | | — |
| ,, 22 | | | — |
| Aug. 5 | | | — |
| ,, 13 | | | — |
| ,, 19 | | | — |

9. *Are We Dealing with one or more than one Species of Trypanosoma?*

As has been shown, no morphological difference can be made out between the trypanosomes found in the cerebro-spinal fluid of cases of Sleeping Sickness, and those found in the blood of persons evidently in good health. Neither can any difference be detected in their behaviour when inoculated into various animals. More experiments are required and more results obtained before this question can be answered unhesitatingly, but at present the evidence distinctly tends to show that we are dealing with one species of Trypanosoma, and that species is probably *Trypanosoma Gambiense,* discovered by Forde and described and named by Dutton.

## 10. *The History and Distribution of Sleeping Sickness in Uganda.*

According to Dr. Moffat, C.M.G.,* Principal Medical Officer, British East Africa and Uganda Protectorates, the first cases of Sleeping Sickness in Uganda were reported by the Drs. Cook, C.M.S. Medical Mission, Mengo, in April, 1901. These cases came from Busoga. On Dr. Moffat's return from leave in April 1902, he was surprised to find that the disease had become epidemic in Busoga, and that it had spread into the neighbouring parts of Uganda and Kavirondo.

He is of opinion that the disease is not indigenous to Uganda, but has been imported during recent times from the Congo basin. This he thinks " is no matter for surprise when we consider that the opening up of Equatorial Africa has led to inter-communication between countries and districts which in earlier days were absolutely cut off from each other." He is inclined to explain the first appearance of the disease in Busoga, by the fact that a large number of Emin Pasha's Soudanese, accompanied by a multitude of slaves from the Congo territories west of Lake Albert, were brought in and settled in Busoga. Dr. Hodges who studied the question in Busoga thinks it first broke out there in 1896. Be this as it may, the disease is now widely spread round the northern shores of the Victoria Nyanza from Buddu on the west, to south of Kavirondo Bay on the east. As far as I am aware the disease has not yet entered German territory.

As the first Sleeping Sickness Commission sent out by the Royal Society made this part of the subject, the distribution of the disease, an object of special investigation, it was thought unnecessary to pursue the subject further. Full details of the work done are not at present available, but for the purposes of this report it may be stated that this disease has a remarkable distribution, being confined to the lake shore and to the islands. How wide the strip of lake shore affected by the disease is, differs in different parts.

Mr. Cubitt, Assistant Collector, Busoga, who investigated the distribution of the disease in his district, in a report to the Commissioner, dated Jinja, June 14, 1902, says " it would seem to be a fairly accurate statement to make that Sleeping Sickness confines itself to the territories adjoining the lake, roughly speaking from a 10 to 20 mile radius of the coast, and that the people living further inland suffer in proportion to the greater or less trading transactions they have with the people living within that radius, while in those who have no trading transactions with the people of the lake there are found no traces of the disease."

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* Foreign Office Despatch, August 5, 1902, Inclosure 2 —
Preliminary Notes on Sleeping Sickness by the Principal Medical Officer, June 17, 1902. Inclosures 3 and 4—Dr. Hodges to Dr. Moffat, May 8, 1902, and May 26, 1902.

In the opinion of the Uganda Prime Minister, Apolo, a strip 10 to 15 miles wide would cover the affected area, and any cases found further inland are imported.

The numerous islands which lie near the northern shores of the lake are also the seat of the disease. Between these islands and the mainland, and for several miles inland a constant interchange of commodities and intercourse between the natives takes place.

There must be some reason for this peculiar distribution. It cannot be due to a food poison, as the natives outside this narrow strip have the same habits. Then, again, if the trypanosomes are the cause of the disease, which there is every reason to suspect, the infection cannot be conveyed in food or clothes, or directly from man to man, but most probably must be carried by some blood-sucking insect.

This led the Commission to ask themselves the following question.

### 11. *Does the Distribution of Sleeping Sickness coincide with the Distribution of any biting Insect?*

On the analogy of Nagana a Tsetse fly was suspected. But the tsetse was not supposed to be present at altitudes of 3000 feet and upwards, nor was it known to be found in Uganda.* A short search in the Botanical Gardens, Entebbe, disposed of these objections. A tsetse fly very similar to the one met with in the fly country, Zululand, was found in numbers. For three months we have had on an average 400 tsetse flies brought into the laboratory every day by a few native children. These flies were caught on the lake shore in the immediate vicinity of Entebbe. This gave a lead. The Prime Minister and Regents were asked to meet the Commission and have the matter explained to them. They at once recognised the fly as one known to the Muganda as Kivu and said that it was found along the shores of the lake.† They were supplied with several dozen nets, killing bottles and boxes, and on their part promised to have the distribution of the fly worked out. A letter was also addressed to the Bishops of Uganda, who kindly promised to obtain information from their Missionaries. Every official and Missionary in the country was then sent a specimen of the tsetse fly, and informed by letter as follows:—

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* Since the above was written I find that Mr. F. J. Jackson in a despatch to the Foreign Office, dated September 27, 1901, writes, "I may add that there is a species of Tsetse fly found along the wooded portion of the lake-shore here at Entebbe. It is plentiful in the Botanical Gardens."

† The tsetse flies found near Entebbe were sent to Professor Ray Lankester, the Director of the British Museum (Natural History), on March 25, and identified by Mr. E. E. Austen as *Glossina palpalis* (Rob. Desv.). He writes: "this is the West African species, and its occurrence in Uganda is very interesting, and was quite unexpected by me, though it is true that another species, *Glossina fusca*, occurs both in West and East Africa."

"Sleeping Sickness in many ways, is very similar to the Fly Disease of South Africa. It is caused by the same kind of parasite, and is possibly carried from man to man by some insect, as the fly disease is carried by the tsetse fly (*Glossina pallidipes*). A species of tsetse fly (*Glossina palpalis*) is also found in Uganda, especially along the shores of the lake and in the islands. It is possible that this fly acts as the carrier of the infective agent of Sleeping Sickness. If this is so, the disease can only occur in places where the fly is found. In other words, no tsetse fly, no Sleeping Sickness.

"In order to settle this question the distribution of this fly must be carefully worked out. To assist in this would you kindly send collections of biting flies from your district and answer the following questions:—

(1) Is the tsetse fly (*Glossina palpalis*) found in your district?

(2) In what kind of place is the fly found—marsh, banana plantation, bank of river, shore of lake, forest, bush or open places?

(3) When does it bite, during the morning, mid-day, or at night?

(4) Is it numerous?

(5) What animals does it bite?

(6) Does Sleeping Sickness occur in the same place, distinguishing of course between imported cases and those which have been infected on the spot?

"The best way to catch the flies is by means of a small butterfly net. They can then easily be killed by a squeeze and placed in a small envelope with the name of the place and date of capture written on it.

"I may add that the natives call the fly Kivu, plural Bevu.

"It is important that the position of the place in which these tsetse flies are found should be exactly laid down. This can be done by giving the latitude and longitude in degrees and minutes, or by saying how many miles it is from a known place in a westerly or S.S.W., etc., direction. Still better would it be to make a tracing of the district, and on this to map out the distribution of the fly and of the sickness.

"It is no use trusting to report. The flies themselves must be caught and sent here for identification. The natives use the word Kivu for several varieties of biting flies.

"If no tsetse flies are found in the district, please send specimen of any of the various biting flies or insects known to the natives."

During June, July, and August some 460 collections of biting flies were sent in from all parts of Uganda. As each package came in the contents were sorted into tsetse flies and other biting flies. If the parcel contained one or more tsetse flies a round red disc was stuck on a map over the locality from which the flies had been sent. If on the other hand no tsetse were found a blue disc was fixed over the spot.

In the same way and at the same time a second map was prepared to show the distribution of Sleeping Sickness. That is to say, if the note on the parcel of flies stated that Sleeping Sickness is prevalent then a red disc was placed over the locality, and if on the contrary no cases of Sleeping Sickness were reported a blue disc was affixed.

Where cases were reported to have been introduced from some other locality, and to have died out without any spread of the disease in the neighbourhood, then a yellow disc was used.

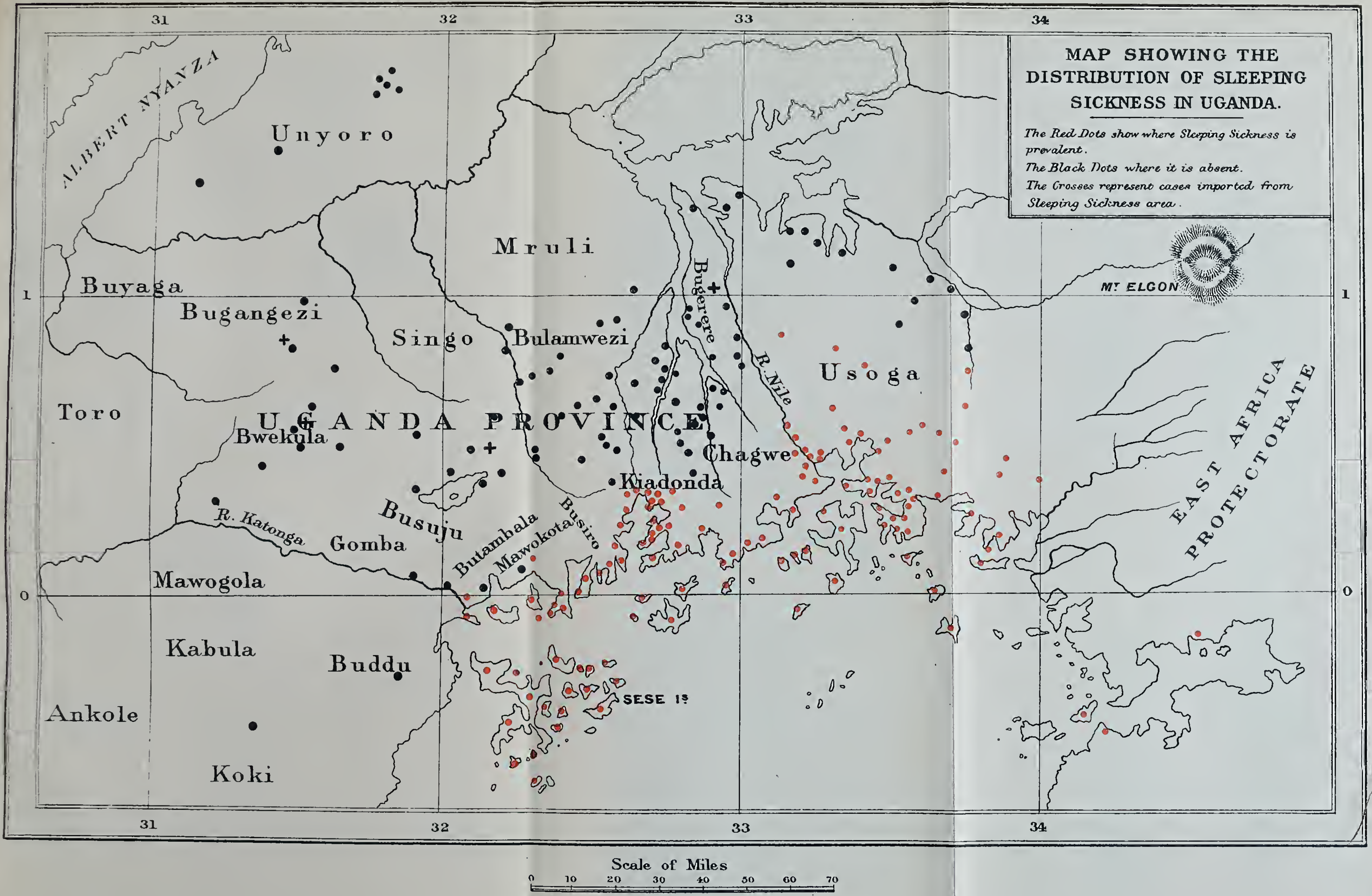
It is evident that two maps so prepared should show at a glance whether the distribution of Sleeping Sickness and *Glossina palpalis* corresponds or not. The accompanying Maps are taken from the originals. They are reduced to one third the size of the larger maps, and the blue discs above mentioned are replaced by black.

On comparing these two maps the similarity of the distribution of Sleeping Sickness and *Glossina palpalis* is self evident. The disease and the fly are limited to the shores of the lake and to the islands, except in Busoga where some of the red dots are seen in the centre of the district. The fly is also seen to pass up the Nile. This is probably due to the fact that the Nile is a great broad waterway, where it issues from the lake at the Ripon Falls, and may be looked upon in its physical characters as an arm of the lake. The other Uganda rivers are mere swamps full of papyrus. As it appeared important to personally examine one of these so-called rivers to discover how far the tsetse fly could be traced up their valleys, one of us (Dr. Nabarro) proceeded on July 4 to explore the Katonga River.

An account of this Expedition is given by Dr. Nabarro as follows :—

"This Expedition proceeded, by canoes, from Entebbe to the Island of Bussi. The tsetse fly was not sought here, as it had been previously found by Colonel Bruce. Then it visited the Islands Zinga and Bunjako, in both of which the tsetse fly was easily found along the shore of the lake where there was forest and short grass. Sleeping Sickness is prevalent on all three islands. Buganga was the next place visited and here, too, the tsetse fly was found as well as large numbers of Tabanidæ. Sleeping Sickness is prevalent in Buganga.

"We left Buganga and sailed round the Katonga Bay to see if there were any channel up the Katonga River but we could find none, the so-called river being a papyrus swamp about 1200 yards wide. We then went further on and landed near Magambo, where forest comes down to the edge of the lake. Here too, the tsetse flies were caught

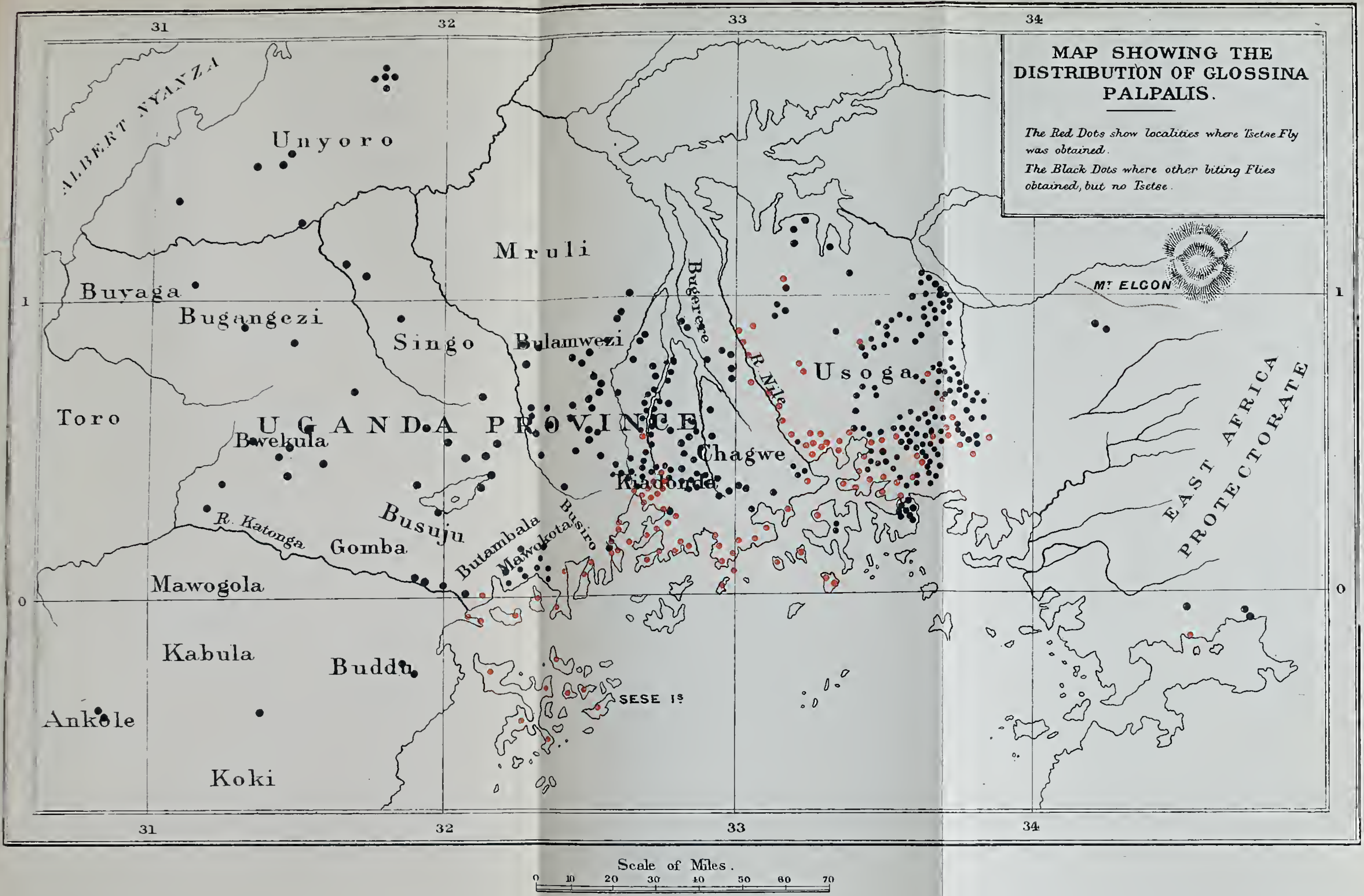


MAP SHOWING THE DISTRIBUTION OF SLEEPING SICKNESS IN UGANDA.

*The Red Dots show where Sleeping Sickness is prevalent.*

*The Black Dots where it is absent.*

*The Crosses represent cases imported from Sleeping Sickness area.*



MAP SHOWING THE DISTRIBUTION OF GLOSSINA PALPALIS.

*The Red Dots show localities where Tsetse Fly was obtained.*

*The Black Dots where other biting Flies obtained, but no Tsetse.*

in numbers and there were several cases of Sleeping Sickness in the neighbourhood.

"From Magambo we marched across the Kibikuta River (1800 yards wide) about 2 miles from where it enters the Victoria Nyanza. No tsetse flies were found there. Thence we marched to the Katonga River, which like the Kibikuta is merely a papyrus swamp. The left bank of the Katonga was explored, from the lake shore, close to the mouth of the river, to a point about 13 or 14 miles up the river and no tsetse flies were seen. So far as I was able to ascertain there is no Sleeping Sickness in this district.

"The return journey to Entebbe was made by road, passing through Metala Maria and Kianjia. In neither of these villages were any tsetse flies found, though Stomoxys and other biting flies exist in large numbers. These two villages are situated about 2—3 miles from the shore of the lake, which here consists of a vast papyrus swamp between the mainland and adjacent Islands Bunjako, Zinga, and Bussi. No tsetse flies were found on the shore close to this swamp. There is no Sleeping Sickness at Mitala Maria or Kianjia.

"From Kianjia we went along the main road to Karabara (5½ miles distant), where we found no tsetse flies and where there is no Sleeping Sickness. We then travelled by the porters' road for 10 miles, *viâ* Busami and Gabongas, and did not find any tsetse flies until we reached the shore of the lake at 'the ferry' near Entebbe. Here many flies were easily caught in the dense forest that there runs along the lake shore. I was informed that there were several cases of Sleeping Sickness in the 'Shambas' near the lake shore, but that there were none in the interior—at Busami or Gabongas."

The main points of interest in Dr. Nabarro's account are that the fly does not proceed up the swampy river valleys and that even on the coast behind papyrus swamps it is also absent.

On the map this journey is shown by a series of red dots on the islands and on the shore at the mouth of the Katonga River, and by blue dots up the river and along the shore behind the islands. The space between the islands and the mainland is one huge papyrus swamp.

In order to work out more minutely the habits of the *Glossina palpalis*, the peninsula on which Entebbe stands was taken in detail and carefully searched for the fly. The result of this showed that the fly is only found on the shore of the lake where there is forest. This forest is thick jungle with high trees and dense undergrowth. The fly is never found on open sandy beaches backed by grass plains, even although there may be some small scrub near the water's edge. It is never found in the grass of the grassy plains, even though the grass be long and tangled. It has not been found by us in banana plantations, nor at any time far from the lake shore.

The habitat then of this fly, in the neighbourhood of Entebbe, is the shore of the lake where there is forest. In Uganda generally the same seems to hold good. In Busoga, on the other hand, it appears to be found inland, but what the physical characters of this province are which would account for this I have not learnt. The fly also passes down the Nile as far as Kakoge Ferry some 50 miles north of the Ripon Falls, and it has even been received from Fajao on the Somerset Nile, and from Tengri and the Achwa River, still further north and near Wadelai.

It is important that the distribution of this fly should be fully worked up, but enough has been done to show that the distribution of this species of tsetse fly is, like Sleeping Sickness, confined to the shores of the lake and the islands. It is on the densely wooded shore of the lake that the half-naked natives of the mainland and islands meet in thousands to trade in fish, bananas, earthenware, etc. If the *Glossina palpalis* can act as a carrier of the trypanosoma of Sleeping Sickness, the circumstances could not be made more favourable than they are for the spread of the disease.

### 12. *Can the Uganda Tsetse Fly* (Glossina palpalis) *Carry the Trypanosoma Found in Sleeping Sickness Cases to Healthy Animals?*

The animal chosen to carry out these experiments was the monkey. These are by far the most suitable animals to work with here. They are easily procured, easily fed, and keep their health perfectly in captivity. The method used was simply to feed tsetse flies on a Sleeping Sickness case and at varying intervals of time to place the same cage of flies on the monkey. The Sleeping Sickness patients did not seem to feel the bites of the flies, as they made no complaint or other signs of inconvenience. There was, as a rule, about 30 flies in each cage, but only those which filled themselves were reckoned as having fed. The following experiments are given in full, as this point is of great importance :—

## EXPERIMENT 114.—Monkey (*Cercopithecus* sp.).

Feeding tsetse flies on a healthy monkey 8 hours after they had been fed on a Sleeping Sickness patient.

| Date. | Number of flies fed on— | | Trypanosomes. |
|---|---|---|---|
| | Patient. | Monkey. | |
| May— | | | |
| 20 | 22 | 0 | Absent |
| 22 | 4 | 5 | |
| 23 | 11 | 2 | |
| 23 | 9 | 7 | |
| 24 | 9 | 6 | |
| 25 | 15 | 1 | |
| 26 | 10 | 4 | |
| 27 | 6 | 5 | |
| 28 | 8 | 7 | Absent |
| 29 | 5 | 4 | |
| 30 | 9 | 3 | |
| 31 | 5 | 1 | |
| June— | | | |
| 1 | 1 | 9 | |
| 2 | 6 | 2 | |
| 3 | 8 | 1 | |
| 4 | 4 | 2 | Absent |
| 5 | 4 | 3 | |
| 6 | 5 | 5 | |
| 7 | 0 | 0 | |
| 8 | 13 | 9 | |
| 9 | 8 | 4 | |
| 10 | 9 | 5 | |
| 11 | 10 | 6 | Absent |
| 12 | 8 | 5 | |
| 13 | 9 | 6 | |
| 14 | 0 | 0 | |
| 15 | 4 | 12 | |
| 16 | 8 | 10 | |
| 17 | 2 | 1 | |
| 18 | 2 | 5 | Absent |
| 19 | 7 | 4 | |
| 20 | 5 | 4 | |
| 21 | 0 | 0 | |
| June— | | | |
| 22 | 16 | 2 | |
| 23 | 24 | 14 | |
| 24 | 31 | 10 | |
| 25 | 25 | 17 | Absent |
| 26 | 28 | 8 | |
| 27 | 17 | 15 | |
| 28 | 0 | 0 | |
| 29 | 26 | 26 | |
| 30 | 24 | 22 | |
| July— | | | |
| 1 | 18 | 15 | Absent |
| 2 | 29 | 16 | |
| 3 | 28 | 11 | |
| 4 | 27 | 14 | |
| 5 | 19 | 13 | |
| 6 | 31 | 16 | |
| 7 | 32 | 11 | |
| 8 | 23 | 13 | |
| 9 | 12 | 15 | |
| 10 | 14 | 0 | |
| 11 | 14 | 28 | |
| 12 | 28 | 6 | |
| 13 | 22 | 18 | |
| 14 | 19 | 12 | |
| 15 | 18 | 8 | |
| 16 | 17 | 13 | |
| 17 | 24 | 9 | Absent |
| 18 | 20 | 8 | |
| 19 | 12 | 11 | |
| 20 | 23 | 17 | |
| 21 | 16 | 13 | |
| 22 | 17 | 11 | 568 |
| 23 | .. | .. | Present |

4 week

### Experiment 115.—Monkey (*Cercopithecus* sp.).

Feeding tsetse flies on a healthy monkey 8 hours after they had been fed on a Sleeping Sickness patient.

| Date. | Number of flies fed on— | | Trypanosomes. |
|---|---|---|---|
| | Patient. | Monkey. | |
| May— | | | |
| 20 | 12 | 0 | Absent |
| 21 | 0 | 3 | |
| 22 | 10 | 2 | |
| 23 | 6 | 1 | |
| 24 | 7 | 2 | |
| 25 | 11 | 5 | |
| 26 | 8 | 4 | |
| 27 | 11 | 1 | |
| 28 | 6 | 3 | |
| 29 | 9 | 8 | Absent |
| 30 | 9 | 4 | |
| 31 | 9 | 3 | |
| June— | | | |
| 1 | 2 | 9 | |
| 2 | 9 | 7 | |
| 3 | 5 | 2 | |
| 4 | 6 | 4 | Absent |
| 5 | 2 | 3 | |
| 6 | 8 | 7 | |
| 7 | 0 | 0 | |
| 8 | 4 | 10 | |
| 9 | 5 | 4 | |
| 10 | 5 | 4 | |
| 11 | 9 | 5 | Absent |
| 12 | 2 | 7 | |
| 13 | 3 | 3 | |
| 14 | 0 | 0 | |
| 15 | 6 | 12 | |
| 16 | 12 | 12 | |
| 17 | 7 | 2 | |
| 18 | 14 | 2 | Absent |
| 19 | 4 | 2 | |
| 20 | 3 | 4 | |
| 21 | 0 | 0 | |

| Date. | Number of flies fed on— | | Trypanosomes. |
|---|---|---|---|
| | Patient. | Monkey. | |
| June— | | | |
| 22 | 29 | 9 | |
| 23 | 18 | 9 | |
| 24 | 21 | 21 | |
| 25 | 23 | 14 | Absent |
| 26 | 17 | 6 | |
| 27 | 16 | 9 | |
| 28 | 0 | 0 | |
| 29 | 33 | 27 | |
| 30 | 28 | 21 | |
| July— | | | |
| 1 | 22 | 17 | Absent |
| 2 | 22 | 19 | |
| 3 | 21 | 13 | |
| 4 | 24 | 10 | |
| 5 | 33 | 10 | |
| 6 | 33 | 10 | |
| 7 | 29 | 12 | |
| 8 | 10 | 16 | |
| 9 | 10 | 12 | |
| 10 | 16 | 13 | |
| 11 | 6 | 18 | |
| 12 | 29 | 8 | |
| 13 | 18 | 11 | |
| 14 | 14 | 9 | |
| 15 | 18 | 12 | |
| 16 | 18 | 7 | |
| 17 | 30 | 7 | Absent |
| 18 | 35 | 17 | |
| 19 | 28 | 8 | |
| 20 | 30 | 8 | |
| 21 | 25 | 7 | |
| 22 | 21 | 14 | |
| 23 | .. | .. | Present |

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9

## Experiment 99. Monkey *Cercopithecus* (sp.).

Feeding tsetse flies on a healthy monkey 24 hours after they had been fed on a Sleeping Sickness patient.

| Date. | Number of flies fed on— | | Trypanosomes. | Date. | Number of flies fed on— | | Trypanosomes. |
|---|---|---|---|---|---|---|---|
| | Patient. | Monkey. | | | Patient. | Monkey. | |
| May— | | | | June— | | | |
| 15 | 16 | | | 19 | .. | 12 | |
| 16 | .. | 7 | Absent | 20 | 15 | | |
| 17 | 6 | | | 21 | .. | 0 | |
| 18 | .. | 6 | | 22 | .. | 11 | |
| 19 | 16 | | | 23 | 21 | | |
| 20 | .. | 3 | | 24 | .. | 23 | |
| 21 | 12 | 2 | | 25 | 26 | .. | Absent |
| 22 | .. | 5 | | 26 | .. | 20 | |
| 23 | 9 | | | 27 | 19 | | |
| 24 | .. | 7 | | 28 | .. | 33 | |
| 25 | 5 | | | 29 | 52 | | |
| 26 | .. | 8 | | 30 | .. | 46 | |
| 27 | 11 | | | | | | |
| 28 | .. | 5 | | July— | | | |
| 29 | 7 | .. | Absent | 1 | 15 | .. | Absent |
| 30 | .. | 9 | | 2 | .. | 20 | |
| 31 | 6 | | | 3 | 31 | | |
| | | | | 4 | .. | 45 | |
| June— | | | | 5 | 27 | | |
| 1 | .. | 7 | | 6 | .. | 27 | |
| 2 | 9 | | | 7 | 40 | | |
| 3 | .. | 6 | | 8 | .. | 17 | |
| 4 | 5 | | | 9 | 15 | | |
| 5 | .. | 4 | Absent | 10 | | 15 | |
| 6 | 8 | | | 11 | 15 | | |
| 7 | .. | 0 | | 12 | .. | 21 | |
| 8 | 10 | | | 13 | 27 | | |
| 9 | .. | 6 | | 14 | .. | 28 | |
| 10 | 17 | | | 15 | 21 | | |
| 11 | .. | 18 | | 16 | .. | 24 | |
| 12 | 18 | | | 17 | 23 | .. | Absent |
| 13 | .. | 17 | | 18 | .. | 23 | |
| 14 | 0 | | | 19 | 36 | | |
| 15 | 8 | | | 20 | .. | 16 | |
| 16 | .. | 11 | | 21 | 26 | | |
| 17 | 0 | | | 22 | .. | 29 | |
| 18 | 10 | .. | Absent | 23 | .. | .. | Present |

10

510

### EXPERIMENT 97. Monkey (*Cercopithecus* sp.).

Feeding tsetse flies on a healthy monkey 24 hours after they had been fed on a Sleeping Sickness patient.

| Date. | Number of flies fed on— | | Trypanosomes. | Date. | Number of flies fed on— | | Trypanosomes. |
|---|---|---|---|---|---|---|---|
| | Patient. | Monkey. | | | Patient. | Monkey. | |
| May— | | | | June— | | | |
| 15 | 11 | .. | Absent | 8 | 28 | | |
| 16 | .. | 1 | | 9 | .. | 16 | |
| 17 | 5 | | | 10 | 17 | | |
| 18 | .. | 8 | | 11 | .. | 24 | Absent |
| 19 | 10 | | | 12 | 23 | | |
| 20 | .. | 5 | | 13 | .. | 20 | |
| 21 | 8 | | | 14 | 0 | | |
| 22 | .. | 9 | Absent | 15 | 11 | | |
| 23 | 13 | | | 26 | .. | 19 | |
| 24 | .. | 8 | | 17 | 14 | | |
| 25 | 6 | | | 18 | 19 | .. | Absent |
| 26 | .. | 9 | | 19 | .. | 7 | |
| 27 | 5 | .. | Absent | 20 | 15 | | |
| 28 | .. | 4 | | 21 | .. | 0 | |
| 29 | 5 | | | 22 | .. | 16 | |
| 30 | .. | 7 | | 23 | 17 | | |
| 31 | 4 | | | 24 | .. | 12 | |
| | | | | 25 | 8 | .. | Absent |
| June— | | | | 26 | .. | 9 | |
| 1 | .. | 1 | | 27 | 23 | | |
| 2 | 4 | | | 28 | .. | 9 | |
| 3 | .. | 5 | | 29 | 26 | | |
| 4 | 5 | .. | Absent | 30 | .. | 56 | |
| 5 | .. | 10 | | | | | |
| 6 | 14 | | | July— | | | |
| 7 | .. | 0 | | 1 | .. | .. | Present |

7 we

155

## EXPERIMENT 116.—Monkey (*Cercopithecus* sp.).

Feeding tsetse flies on a healthy monkey 48 hours after they had been fed on a Sleeping Sickness patient.

| Date. | Number of flies fed on— | | Trypanosomes. |
|---|---|---|---|
| | Patient. | Monkey. | |
| May— | | | |
| 20 | 10 | .. | Absent |
| 21 | | | |
| 22 | .. | 10 | |
| 23 | | | |
| 24 | 9 | | |
| 25 | | | |
| 26 | .. | 5 | |
| 27 | | | |
| 28 | 9 | | |
| 29 | | | |
| 30 | .. | 6 | Absent |
| 31 | | | |
| June— | | | |
| 1 | 6 | | |
| 2 | | | |
| 3 | .. | 7 | |
| 4 | .. | .. | Absent |
| 5 | 11 | | |
| 6 | | | |
| 7 | .. | 4 | |
| 8 | | | |
| 9 | 6 | | |
| 10 | | | |
| 11 | .. | 7 | Absent |
| 12 | | | |
| 13 | 16 | | |
| 14 | | | |
| 15 | .. | 8 | |
| 16 | | | |
| 17 | 17 | | |
| 18 | 11 | .. | Absent |
| 19 | | | |
| 20 | .. | 3 | |
| 21 | | | |
| 22 | 19 | | |
| 23 | | | |
| 24 | .. | 15 | |
| 25 | .. | .. | Absent |
| 26 | 26 | | |
| 27 | | | |
| 28 | .. | 16 | |
| 29 | 9 | | |
| 30 | | | |
| July— | | | |
| 1 | .. | 49 | |
| 2 | | | |
| 3 | 51 | .. | Absent |
| 4 | | | |
| 5 | .. | 29 | |
| 6 | | | |
| 7 | 40 | | |
| 8 | | | |
| 9 | .. | 20 | |
| 10 | | | |
| 11 | 25 | | |
| 12 | | | |
| 13 | .. | 26 | |
| 14 | | | |
| 15 | 25 | | |
| 16 | | | |
| 17 | .. | 16 | Absent |
| 18 | | | |
| 19 | 64 | | |
| 20 | | | |
| 21 | .. | 46 | |
| 22 | | | |
| 23 | .. | .. | Present |

415

267

As the result of these five experiments it may be considered proved that *Glossina palpalis* can convey trypanosomes from Sleeping Sickness cases to healthy monkeys up to at least 48 hours after feeding. We have seen that every third or fourth native in the Sleeping Sickness area carries trypanosomes in his blood, so that the fly has plenty of material to draw from. It may be objected that the parasites are so scarce in the blood that it is unlikely the fly will get hold of them. The same objection might be urged in regard to Nagana,

91

whose trypanosoma is probably scarcer in the blood of the wild animals than is the case in Sleeping Sickness. No doubt cases do occur among Sleeping Sickness patients, though rarely, in which the trypanosomes are fairly numerous. It is thought that they may be more numerous in the early stages when still confined to the blood than in the later stage of Sleeping Sickness. The observations on this point are as yet neither numerous or exact enough to warrant any dogmatic statement.

The next series of experiments was made to find out if the ordinary wild tsetse flies, freshly caught and placed on healthy monkeys without any artificial feeding, will give rise to the disease.

This is a still more important point, and in this also the experiments are given in full. The flies were caught on the shore of the lake in the vicinity of Entebbe, and when brought to the laboratory were immediately placed on healthy monkeys.

### 13. *Can Freshly-Caught* Glossina palpalis *convey Trypanosomes to Healthy Animals?*

EXPERIMENT 94.—Monkey (*Cercopithecus* sp.).

To ascertain if tsetse flies, freshly caught in the vicinity of Entebbe, are carrying trypanosomes.

May 13, 1903. Blood examined. No trypanosomes. No malaria.

| | |
|---|---|
| May 13......... | Fed 31 flies freshly caught near Entebbe. |
| „ 15......... | „ 15 „ |
| „ 18......... | „ 10 „ |
| „ 19......... | „ 20 „ |
| „ 20......... | „ 13 „ |
| „ 21......... | „ 16 „ |

Blood examined. Trypanosoma absent. Malaria absent.

2 weeks

215

| | |
|---|---|
| May 22......... | Fed 20 flies. |
| „ 23......... | „ 25 „ |
| „ 24......... | „ 17 „ |
| „ 25......... | „ 31 „ |
| „ 26......... | „ 18 „ |
| „ 27......... | |

Blood examined. Trypanosomes *present*. Malaria absent.

EXPERIMENT 130. Monkey (*Cercopithecus* sp.).

To note the effect of feeding freshly-caught tsetse flies on a healthy monkey.

| | |
|---|---|
| June 10, 1903. | Fed 60 freshly-caught flies. |
| „ 11..... ... | „ 23 flies. |

Blood examined. Trypanosomes absent. Malaria present.

| | | | | |
|---|---|---|---|---|
| June | 12......... | Fed | 12 | flies. |
| ,, | 13......... | ,, | 26 | ,, |
| ,, | 14......... | ,, | 20 | ,, |
| ,, | 17......... | ,, | 25 | ,, |
| ,, | 18......... | ,, | 10 | ,, |

Blood examined. Trypanosomes absent. Malaria present.

| | | | | |
|---|---|---|---|---|
| June | 19......... | Fed | 9 | flies. |
| ,, | 20......... | ,, | 19 | ,, |
| ,, | 21......... | ,, | 30 | ,, |
| ,, | 22......... | ,, | 7 | ,, |
| ,, | 23......... | ,, | 74 | ,, |
| ,, | 24......... | ,, | 31 | ,, |
| ,, | 25......... | ,, | 28 | ,, |

Blood examined. Trypanosomes absent. Malaria present.

| | | | | |
|---|---|---|---|---|
| June | 26......... | Fed | 83 | flies. |
| ,, | 27......... | ,, | 64 | ,, |
| ,, | 28......... | ,, | 27 | ,, |
| ,, | 29......... | ,, | 38 | ,, |
| ,, | 30......... | ,, | 62 | ,, |
| July | 1......... | ,, | 62 | ,, |

Blood examined. Trypanosomes absent. Malaria present.

| | | | | |
|---|---|---|---|---|
| July | 2......... | Fed | 140 | flies. |
| ,, | 3......... | ,, | 35 | ,, |
| ,, | 4......... | ,, | 34 | ,, |
| ,, | 5......... | ,, | 23 | ,, |
| ,, | 6......... | ,, | 44 | ,, |
| ,, | 7......... | ,, | 14 | ,, |
| ,, | 8......... | ,, | 34 | ,, |
| ,, | 9......... | | | |

4½

Examined blood. Trypanosomes *present*. Malaria present.

EXPERIMENT 131. Monkey. (*Cercopithecus* sp.)

Feeding freshly-caught tsetse flies on a healthy monkey.

| | | | | |
|---|---|---|---|---|
| July | 17......... | Fed | 5 | flies. |
| ,, | 18......... | ,, | 3 | ,, |
| ,, | 19......... | ,, | 7 | ,, |
| ,, | 20......... | ,, | 17 | ,, |

Examination of blood. Trypanosomes absent. Malaria present.

32

| | |
|---|---|
| June 21......... | Fed 23 flies. |
| ,, 22......... | ,, 7 ,, |
| ,, 23......... | ,, 28 ,, |
| ,, 24...... .. | ,, 39 ,, |
| ,, 25......... | ,, 13 ,, |

Examination of blood. Trypanosomes absent. Malaria present.

| | |
|---|---|
| June 26......... | Fed 53 flies. |
| ,, 27......... | ,, 47 ,, |
| ,, 28......... | ,, 5 ,, |
| ,, 29......... | ,, 17 ,, |
| ,, 30......... | ,, 108 ,, |
| July 1......... | ,, 36 ,, |

Examination of blood. Trypanosomes absent. Malaria present.

| | |
|---|---|
| July 2......... | Fed 98 flies. |
| ,, 3......... | ,, 45 ,, |
| ,, 4.. ...... | ,, 28 ,, |
| ,, 6......... | ,, 38 ,, |
| ,, 7......... | ,, 17 ,, |
| ,, 8......... | ,, 36 ,, |
| ,, 9......... | ,, 57 ,, |
| ,, 10......... | ,, 30 ,, |

956

Examination of blood. Trypanosomes *present*. Malaria present.

These important experiments have succeeded quicker than we anticipated.

The flies were caught in the vicinity of the hut-tax labourers' camp. These men to the number of thousands come to Entebbe and work for Government for one month in lieu of paying hut-tax. They live in rudely built grass huts near the shores of the lake, about a mile from Entebbe, along the Kampala Road. Tsetse flies caught at this place and brought in cages to the laboratory have conveyed trypanosomes to three monkeys. What could be more suggestive? It is true that the trypanosoma, conveyed to these monkeys, may be a different species from that found in Sleeping Sickness, but taking everything into consideration, the probabilities are that it is the same.

If this is so it would be interesting and instructive to know what percentage of the hut-tax labourers develop this disease on their return to their homes. It may possibly be an extremely small figure, as these men are only exposed to the risk for a month, and, as a rule, their work lies in the town where tsetse flies are never found. This is very different to the case of the natives, who live all their lives on the edge of the lake and are exposed all the year round to the infection. In our opinion if these hut-tax labourers were camped on suitable ground, there would be little or no danger in their coming into Entebbe to do a month's work.

Up to the present we have received very few notices of the occurrence of cases of Sleeping Sickness, in Non-Sleeping Sickness areas, supposed to have been contracted in Entebbe or on the lake shore.

Five cases are reported by the White Fathers to have occurred at Kiambalangos Place, south of Hoima, and three cases in Bwekula* by Mr. Lewin. These men are stated to have contracted the disease round about the Victoria Lake, and have all died out without spreading the disease. Another curious case is reported by Major Harrison, K.A.R., at Nairobi, in British East Africa. The man was a bugler named Mahomed. He left Uganda in May, 1900, enlisted at Nairobi, British East Africa, on November 7th. 1900, and died of Sleeping Sickness February 18, 1902. Dr. Wiggins also relates the case of an Askari or native policeman named Sulivan Khair, stationed at Fort Ternan, British East Africa, for 2½ years before he developed the disease. He came from Entebbe, and except for a visit to Jinja, Busoga, 14 months before he died, he had not left Fort Ternan. Neither of these men set up infection among their comrades.

These cases hint at a long incubation period when the trypanosoma is confined to the blood, and also to the need of the tsetse fly to act as intermediary between the sick and healthy.

## APPENDIX.

In the Progress Report several brief histories of cases were given. To these are added the following:—

CASE 28.—Kagoya. Female. Age 20 Years. District, Bukola, in Busoga. Food, Bananas, Sweet Potatoes, and Fish.

November 6, 1902. Admitted to hospital. Patient states that one sister died of Sleeping Sickness.

---

* 1. Middle-aged man, two years in Chagwe, came to Bwekula ill, and died in about six months. In 1902.

2. Middle-aged man, one year round about Entebbe; came back ill; died in about four months. 1903.

3. Young man, trading near Victoria Lake six months; came back ill; died in about three months. 1903.

In all three cases no one suffered from Sleeping Sickness in the vicinity through contact with these.

March 27, 1903. She has been ill 7 or 8 months. The chief symptoms now are tremor, weakness, and peculiar speech.

April 7. *General Condition.*—The girl is very well nourished, in fact quite plump. She stands with difficulty, and for the last few days has been unable to walk without assistance. She seems to be quite intelligent, but her face is dull, listless, and expressionless, and she stares about in a vacant way with her eyes wide open. There is no œdema of any part of the body. The colour of the mucous membranes is normal, and there are no trophic changes. Lymphatic glands are generally enlarged, in the anterior triangles being about the size of peas, and in the inguinal region of beans. She complains of pain in the lower extremities. Her pulse is 100°. Her temperature subnormal. In regard to cutaneous eruptions, a few small ulcers can be seen on the chest, right arm and back.

*Nervous System.*—Her intelligence and memory are good. She is reported to sleep well at night and also a good deal during the day. Her speech is slow, tremulous. indistinct, faint and high pitched like a whimpering child. There is nothing noteworthy about her eyes. There is marked tremor of the tongue, but not of the lips or hands. Her muscular nutrition is very good, she is sensible to touch, temperature and pain, and her muscle sense is normal. In regard to her reflexes, the knee jerk is diminished, and the ankle clonus is present to a slight extent.

*Alimentary System.*—Her appetite is reported to be good, her tongue is moist, covered with white fur and indented by the teeth, otherwise there is nothing abnormal.

*Circulatory System.*—The apex beat is imperceptible, and on auscultation the heart sounds are weak, regular, and there is no bruit. The pulse has a rate of 100, with low tension, small size, easily compressible, and regular rhythm.

*Respiratory System.*—Nothing noteworthy.

*Cutaneous System.*—The skin is harsh and rough, and the papillæ are very prominent, especially over the legs and arms.

*Eyes.*—On ophthalmoscopic examination both found to be normal.

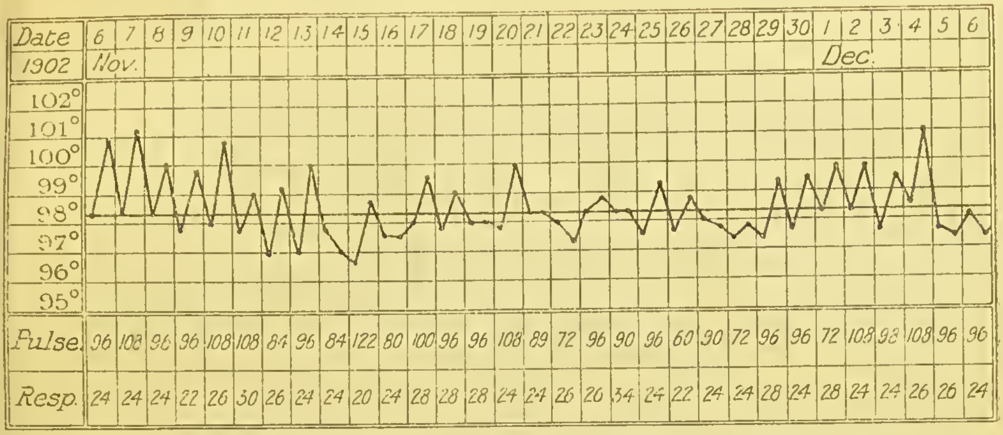
April 20. Patient is now very tremulous, the tremors of the tongue being marked. The heart action is feeble.

May 6. Patient has been bed-ridden for the last month. She lies all day in a lethargic condition.

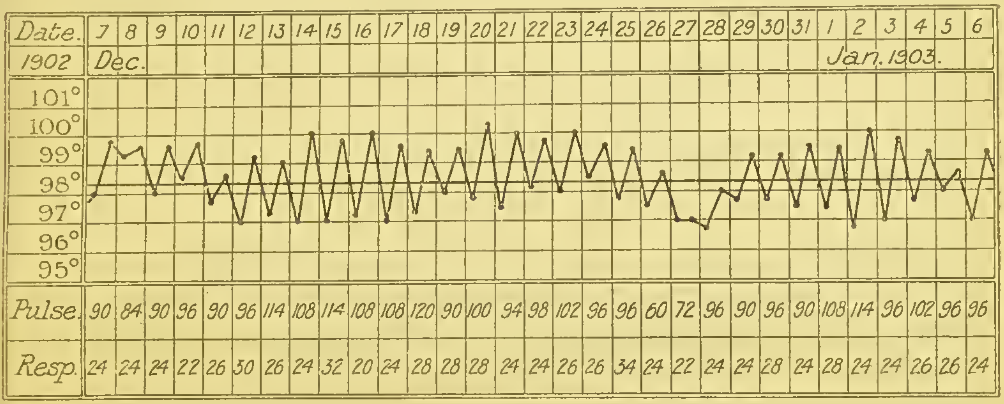
May 28. Gradually getting feebler. She is still well nourished, and is able to sit up in bed.

June 5. Condition distinctly worse to-day. She is practically unconscious and her breathing is stertorous. Died 9 P.M.

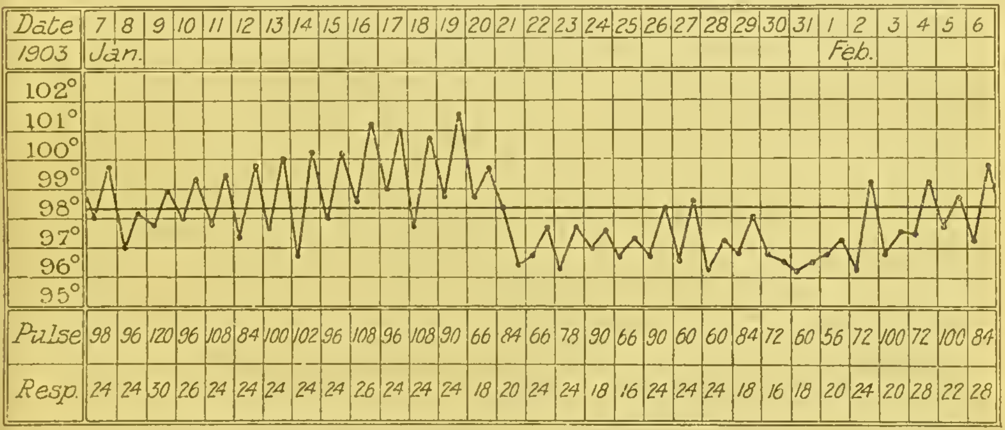
The following chart represents the course of the disease :—



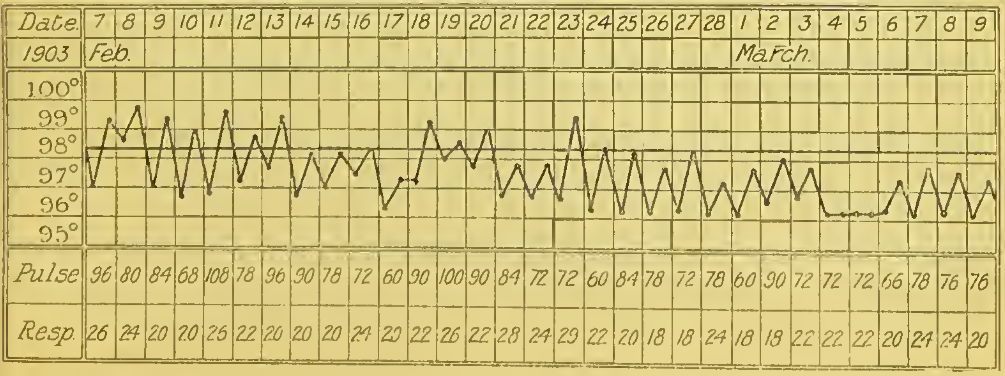
| Date | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1902 | Nov. | | | | | | | | | | | | | | | | | | | | | | | | | Dec. | | | | | |
| Pulse | 96 | 108 | 96 | 96 | 108 | 108 | 84 | 96 | 84 | 122 | 80 | 100 | 96 | 96 | 108 | 89 | 72 | 96 | 90 | 96 | 60 | 90 | 72 | 96 | 96 | 72 | 108 | 98 | 108 | 96 | 96 |
| Resp. | 24 | 24 | 24 | 22 | 26 | 30 | 26 | 24 | 24 | 20 | 24 | 28 | 28 | 28 | 24 | 24 | 26 | 20 | 34 | 24 | 22 | 24 | 24 | 28 | 24 | 28 | 24 | 24 | 26 | 26 | 24 |



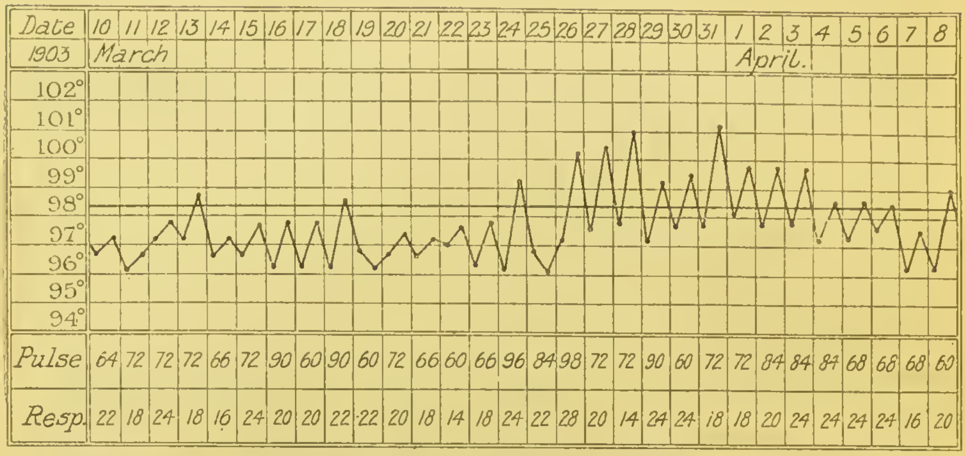
| Date. | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1902 | Dec. | | | | | | | | | | | | | | | | | | | | | | | | | Jan. 1903. | | | | | |
| Pulse. | 90 | 84 | 90 | 96 | 90 | 96 | 114 | 108 | 114 | 108 | 108 | 120 | 90 | 100 | 94 | 98 | 102 | 96 | 96 | 60 | 72 | 96 | 90 | 96 | 90 | 108 | 114 | 96 | 102 | 96 | 96 |
| Resp. | 24 | 24 | 24 | 22 | 26 | 30 | 26 | 24 | 32 | 20 | 24 | 28 | 28 | 28 | 24 | 24 | 26 | 26 | 34 | 24 | 22 | 24 | 24 | 28 | 24 | 28 | 24 | 24 | 26 | 26 | 24 |

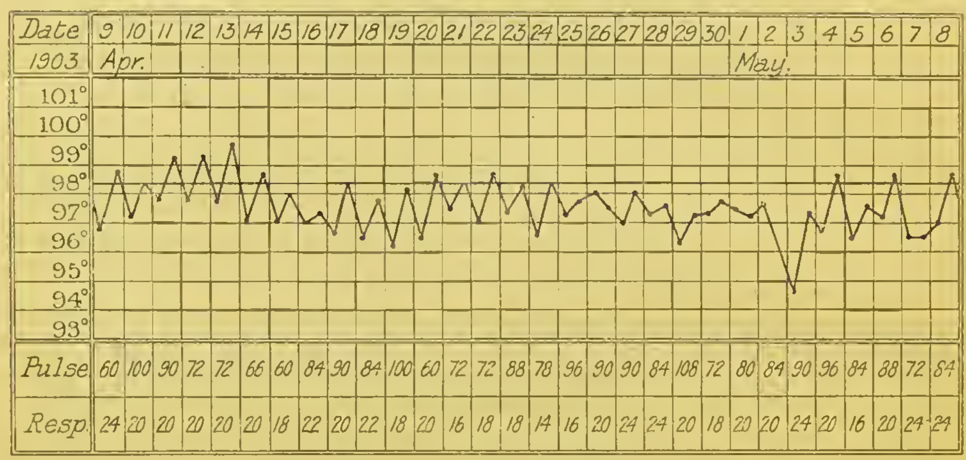


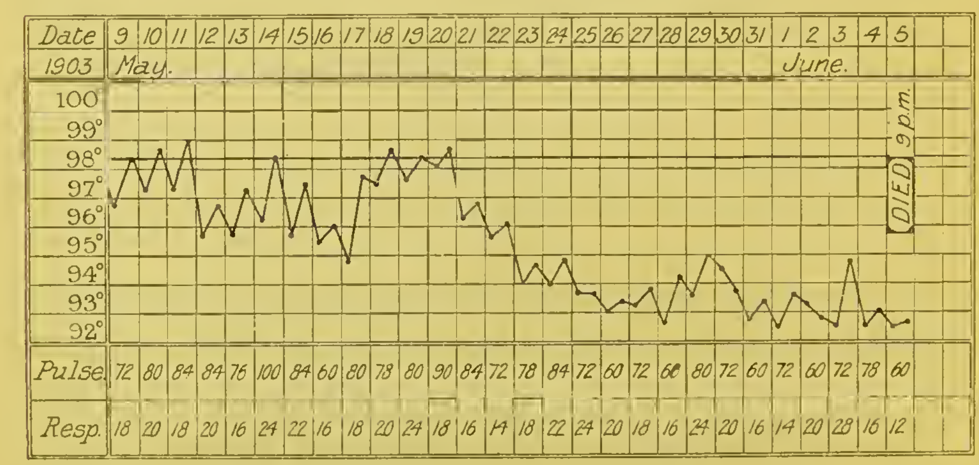
| Date | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | Jan. | | | | | | | | | | | | | | | | | | | | | | | | | Feb. | | | | | |
| Pulse | 98 | 96 | 120 | 96 | 108 | 84 | 100 | 102 | 96 | 108 | 96 | 108 | 90 | 66 | 84 | 66 | 78 | 90 | 66 | 90 | 60 | 60 | 84 | 72 | 60 | 56 | 72 | 100 | 72 | 100 | 84 |
| Resp. | 24 | 24 | 30 | 26 | 24 | 24 | 24 | 24 | 24 | 26 | 24 | 24 | 24 | 18 | 20 | 24 | 24 | 18 | 16 | 24 | 24 | 24 | 18 | 16 | 18 | 20 | 24 | 20 | 28 | 22 | 28 |



| Date. | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | Feb. | | | | | | | | | | | | | | | | | | | | | | March. | | | | | | | | |
| Pulse | 96 | 80 | 84 | 68 | 108 | 78 | 96 | 90 | 78 | 72 | 60 | 90 | 100 | 90 | 84 | 72 | 72 | 60 | 84 | 78 | 72 | 78 | 60 | 90 | 72 | 72 | 72 | 66 | 78 | 76 | 76 |
| Resp. | 26 | 24 | 20 | 20 | 25 | 22 | 20 | 20 | 20 | 24 | 20 | 22 | 26 | 22 | 28 | 24 | 29 | 22 | 20 | 18 | 18 | 24 | 18 | 18 | 22 | 22 | 22 | 20 | 24 | 24 | 20 |


Date 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8
1903 March April.
102° 101° 100° 99° 98° 97° 96° 95° 94°
Pulse 64 72 72 72 66 72 90 60 90 60 72 66 60 66 96 84 98 72 72 90 60 72 72 84 84 84 68 68 68 60
Resp. 22 18 24 18 16 24 20 20 22 22 20 18 14 18 24 22 28 20 14 24 24 18 18 20 24 24 24 24 16 20


Date 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8
1903 Apr. May.
101° 100° 99° 98° 97° 96° 95° 94° 93°
Pulse 60 100 90 72 72 66 60 84 90 84 100 60 72 72 88 78 96 90 90 84 108 72 80 84 90 96 84 88 72 84
Resp. 24 20 20 20 20 20 18 22 20 22 18 20 16 18 18 14 16 20 24 24 20 18 20 20 24 20 16 20 24 24


Date 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5
1903 May. June.
100° 99° 98° 97° 96° 95° 94° 93° 92°
DIED 9 p.m.
Pulse 72 80 84 84 76 100 84 60 80 78 80 90 84 72 78 84 72 60 72 60 80 72 60 72 60 72 78 60
Resp. 18 20 18 20 16 24 22 16 18 20 24 18 16 14 18 22 24 20 18 16 24 20 16 14 20 28 16 12

The following table shows the presence or absence of trypanosomes in the cerebro-spinal fluid and blood:—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 27 .... | | | | | + |
| Apr. 9 .... | + | – | – | | + |
| Apr. 22 .... | | – | | | |

June 6, 1903. *Post-mortem.*—14 hours after death.

The body is that of a well-nourished woman. No cutaneous eruptions or bed-sores present. Superficial lymphatic glands only slightly enlarged. Rigor mortis present.

*Brain.*—Dura mater healthy, but bulging slightly a little to the right of the longitudinal line, about 1 inch behind the coronal suture; this was due to dilated vessels. On cutting through the dura mater, the surface of the brain is seen to be congested, the vessels considerably dilated, and some of them containing decolourised thrombi. The brain looks dry; opaque and glassy-looking, a typical Sleeping Sickness brain, with increase of sub-arachnoid fluid and flattening of convolutions. On section the brain is normal throughout. Weight 2 lb. 10 oz.

Body opened in the usual way. There is a thick layer of subcutaneous fat all over the body. No free fluid in pericardial, pleural or peritoneal cavities.

*Heart.*—Excess of fat, especially about the right ventricle. Orifices and valves healthy. Muscular substance normal. No petechiæ. Weight 8 oz.

*Lungs.*—Left, no adhesions. Weighs 9 oz. Lower lobe slightly congested, otherwise normal. Right, no adhesions. Weighs 9 oz. Marked congestion of lower lobe and slight congestion of upper lobe.

Mediastinal and bronchial glands not enlarged.

*Spleen.*—Weight 7 oz. Substance firm. No excess of fibrous tissue. No pigmentation.

*Liver.*—Right lobe slightly adherent to diaphragm. Weight 2 lb. 9 oz. Looks healthy. Moderately firm.

*Kidneys.*—Each weighs 2 oz. Healthy on section. Capsules strip off readily. *Intestines.*—Healthy. *Mesenteric glands*, not enlarged, but much fat present.

*Remarks.*—This is a typical uncomplicated case of Sleeping Sickness, remarkable for its duration, the patient having been under observation in hospital for 7 months. At death there was no emaciation, on the contrary the girl remained plump and well nourished. The trypanosoma was not found in the blood, but only one examination was made.

CASE 20.—Seera (Male). Age 25 years. District Bussi. Occupation, Canoe man.

March 24, 1903. Admitted to hospital. Patient states that there had been no cases of Sleeping Sickness in the same house, and that he had been ill about 6 months. The first symptoms were weakness, then drowsiness.

April 7. Patient has a heavy look. His speech is weak, low, slow and indistinct.

April 17. The chief symptoms now are weakness, a heavy look, a weak, mumbling, indistinct and slow speech, and tremors of tongue, lips and the muscles of the limbs and trunk.

April 18. *Physical examination.*—Development and nutrition good. Gait weak and slightly shuffling. Intelligence fair. Expression of face, dull. There is a slight œdematous patch over tibia, probably due to chiggers. Lympahtic glands enlarged. No pain complained of. Pulse 90, weak and regular. Some pityriasis versicolour.

*Nervous System.*—Intelligence good. Memory good. Sleep; lies about all day, but does not sleep much.

*Eyes.*—No abnormality. Pupils respond to light.

Tremor of tongue marked, lips slight, hands marked. He has an involuntary movement of the right forefinger and right thumb as if trying to rub something off the thumb, or as if he were twisting a thread.

Reflexes.—Knee jerks increased. Elbow and wrist jerks also increased.

*Alimentary System.*—Has a good appetite. Tongue moist, slightly furred. Liver not enlarged. Spleen not palpable.

*Heart.*—Normal. Pulse 90, tension low. Size small, compressible and regular.

*Respiratory System.*—Nothing noteworthy.

*Cutaneous System.*—Nothing abnormal.

May 6. Patient's condition remains about the same. He often has a considerable rise of temperature at night. The blood has been examined five times with the centrifuge for trypanosomes, but unsuccessfully. He is in the second stage of the disease.

May 13. Patient is about the same. Tremor of tongue and lips. He has slight attacks of fever with rise of temperature at night. His gait and general nutrition still good.

May 29. Patient is still well nourished. His speech is slow, and expression dull.

June 3. General condition shows no alteration.

June 10. Examined blood by puncturing spleen. No trypanosomes.

" 16. Patient appears more drowsy and dull.

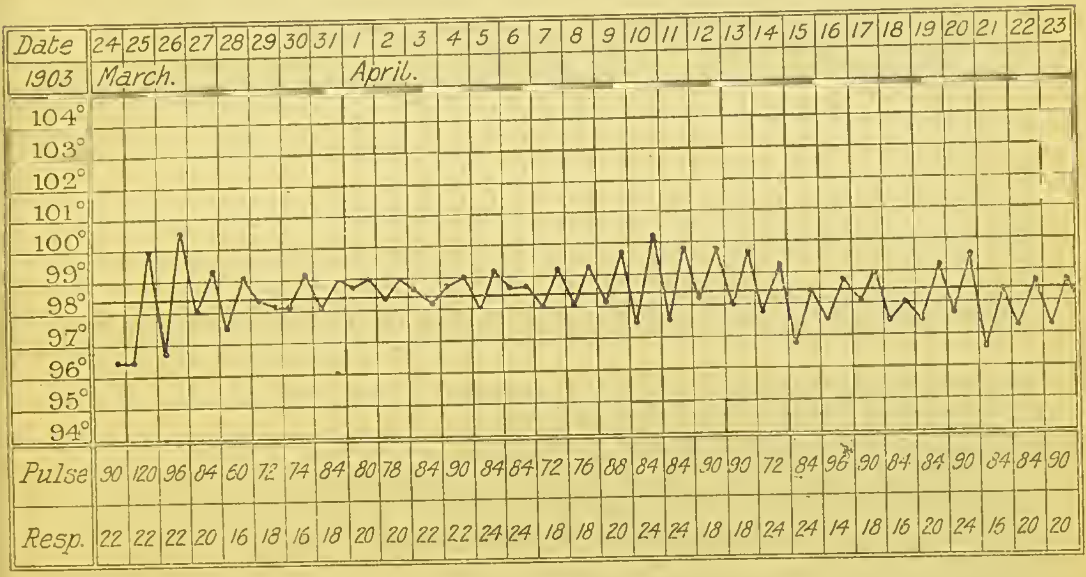
" 20. Patient is getting more and more bed-ridden.

" 24. Condition is more pronounced.

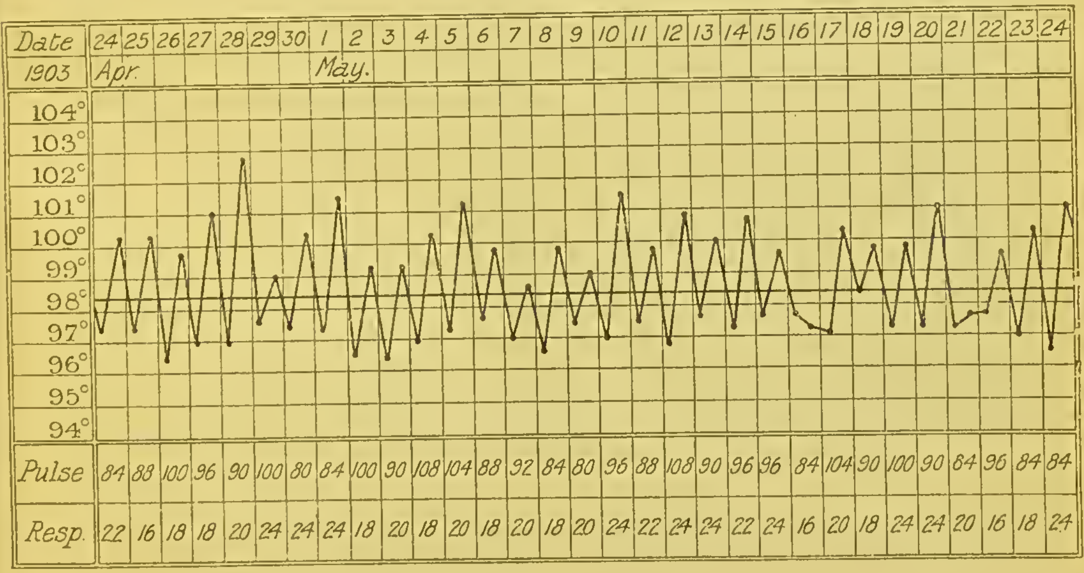
" 28. Patient appears to be distinctly in the third stage of the disease.

July 7. Died 3 P.M. No *post-mortem*.

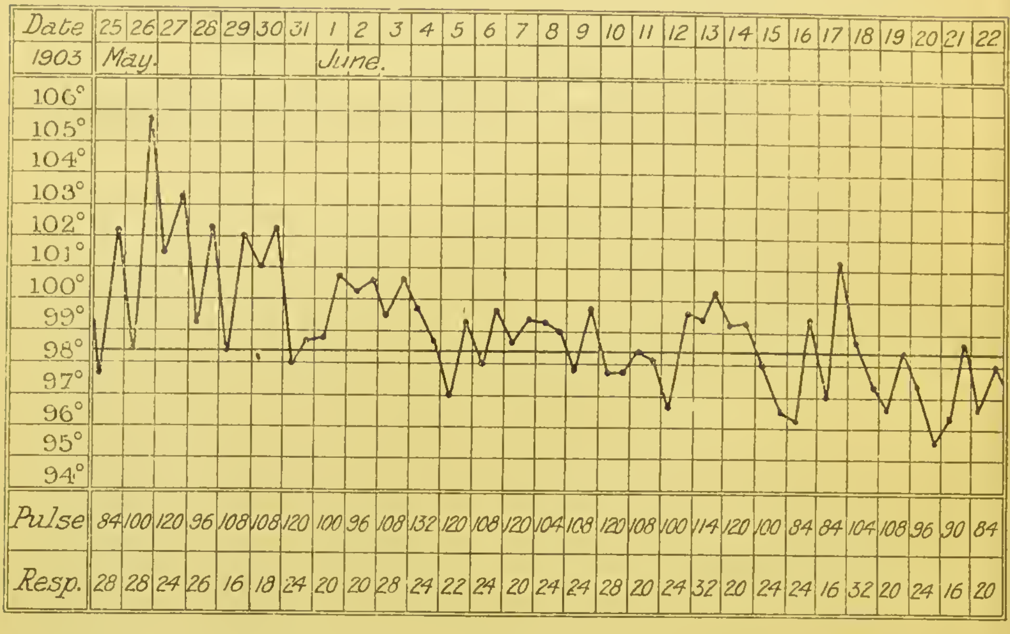
The following chart represents the course of the disease :—

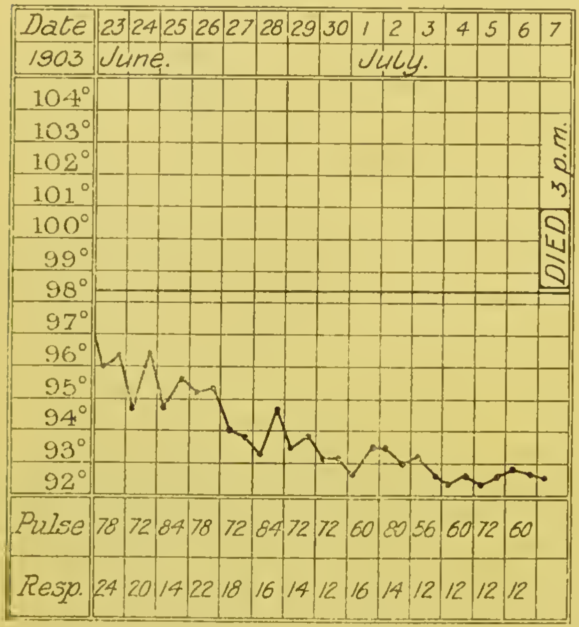


| Date | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | March. | | | | | | | | April. | | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 90 | 120 | 96 | 84 | 60 | 72 | 74 | 84 | 80 | 78 | 84 | 90 | 84 | 84 | 72 | 76 | 88 | 84 | 84 | 90 | 90 | 72 | 84 | 96 | 90 | 84 | 84 | 90 | 84 | 84 | 90 |
| Resp. | 22 | 22 | 22 | 20 | 16 | 18 | 16 | 18 | 20 | 20 | 22 | 22 | 24 | 24 | 18 | 18 | 20 | 24 | 24 | 18 | 18 | 24 | 24 | 14 | 18 | 16 | 20 | 24 | 16 | 20 | 20 |



| Date | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | Apr. | | | | | | | May. | | | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 84 | 88 | 100 | 96 | 90 | 100 | 80 | 84 | 100 | 90 | 108 | 104 | 88 | 92 | 84 | 80 | 96 | 88 | 108 | 90 | 96 | 96 | 84 | 104 | 90 | 100 | 90 | 84 | 96 | 84 | 84 |
| Resp. | 22 | 16 | 18 | 18 | 20 | 24 | 24 | 24 | 18 | 20 | 18 | 20 | 18 | 20 | 18 | 20 | 24 | 22 | 24 | 24 | 22 | 24 | 16 | 20 | 18 | 24 | 24 | 20 | 16 | 18 | 24 |


Date
1903
25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22
May.
June.
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
94°
Pulse
84 100 120 96 108 108 120 100 96 108 132 120 108 120 104 108 120 108 100 114 120 100 84 84 104 108 96 90 84
Resp.
28 28 24 26 16 18 24 20 20 28 24 22 24 20 24 24 28 20 24 32 20 24 24 16 32 20 24 16 20


Date
1903
23 24 25 26 27 28 29 30 1 2 3 4 5 6 7
June.
July.
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
94°
93°
92°
DIED 3 p.m.
Pulse
78 72 84 78 72 84 72 72 60 80 56 60 72 60
Resp.
24 20 14 22 18 16 14 12 16 14 12 12 12 12

The following table shows the presence or absence of trypanosomes in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 26.... | | | | | + |
| Apr. 9.... | – | | – | | |
| „ 17.... | + | | – | | |
| „ 21.... | + | | – | | |
| „ 22.... | | – | | | |
| „ 27.... | + | – | – | | |
| „ 30.... | | – | | | |
| May 5.... | | | – | | |
| „ 13.... | + | – | – | | |
| „ 21.... | + | – | – | | – |
| „ 30.... | + | – | – | | + |
| June 10.... | | – | – | | |

*Remarks.*—This is an ordinary uncomplicated case of Sleeping Sickness of long duration.

The remarkable point in it is that in spite of repeated examinations, the trypanosomes were never found in the blood. In the series of cases taken to show the presence or absence of the parasites in the blood, this was the only case which gave a negative result. Even puncturing the spleen and examining the pulp showed nothing.

### CASE 13.—Esaka. Male. Age 25 Years.

March 25. Admitted to hospital.

April 4. *General Condition.*—State of development and nutrition good. Gait all right. Intelligence good. Expression of face bright, cheerful and smiling. No œdema. Lymphatic glands generally enlarged, slight to marked. Complains of frontal headache. Breathes normally. Pulse 100, normal. Temperature 102. No cutaneous eruptions.

*Nervous System.*—Intelligence good. Memory good. Sleeps well. No change in speech. Eyes normal.

Motor functions. Tremors. Tongue not marked, lips none, hands none. Muscular nutrition good. Sensory functions normal.

Reflexes. Knee jerk normal. No ankle clonus.

*Alimentary System.*—Appetite good. Tongue furred, moist. Teeth good. Abdomen normal. Stomach normal in size. Liver not enlarged. Spleen not palpably enlarged.

*Circulatory System.*—Heart. Apex beat between fourth and fifth ribs, ½ inch to right of nipple, not enlarged. Auscultation. Normal but distant. No bruits. Pulse 100. Tension good, size good, compression medium, rhythm regular.

*Respiratory System.*—Nothing noteworthy.

*Skin.*—The skin is normal, and there are no eruptions.

May 6. Patient is not so cheerful as on first coming into hospital. There is a slight tremor of the tongue, but none of fingers. His expression is wistful and rather miserable. His speech is good.

May 13. He still walks well and his speech is normal. He sleeps a good deal during the day.

May 16. Patient is getting rapidly worse. He remains in bed the greater part of the day. Tremor of tongue very marked.

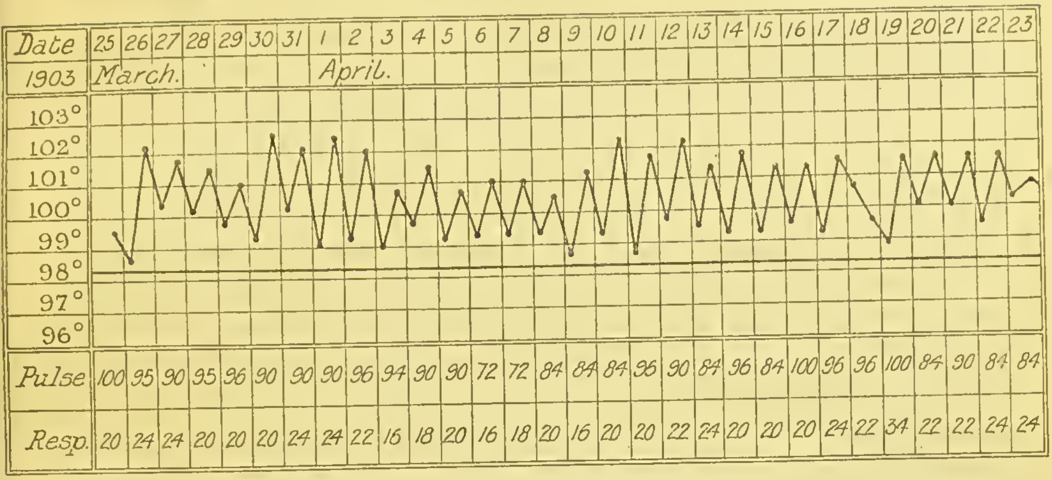
May 20. The temperature has been lower during the past week, and yesterday fell to 95° F. Marked tremors of tongue, but none of fingers. His gait is very hesitating. Expression wistful. Voice weak.

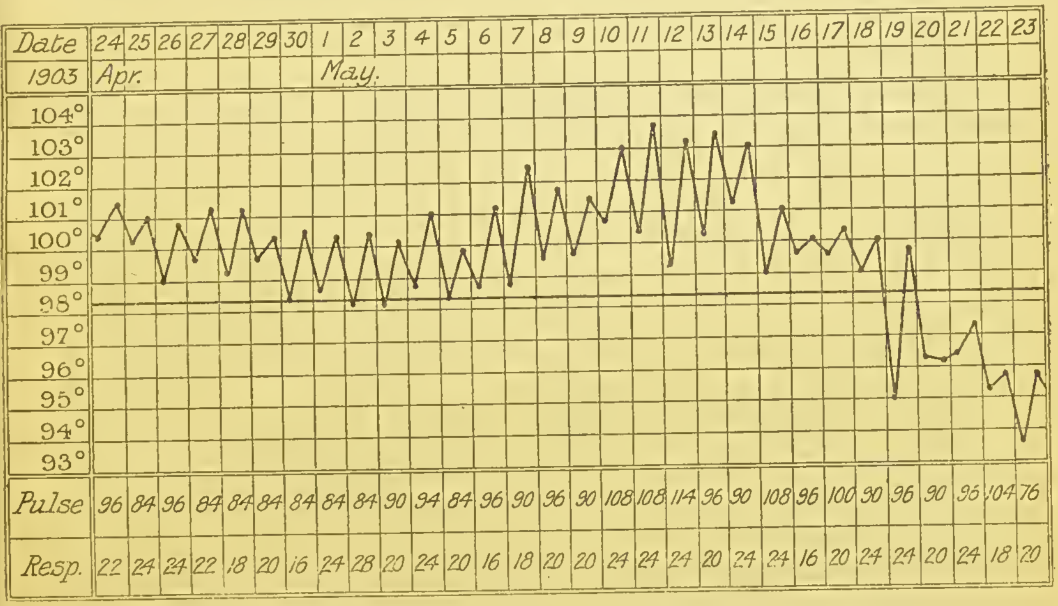
May 24. Patient is rapidly going down hill. This morning he is lying in bed in a dazed, semi-conscious condition, with his eyes wide open. When told to put out his tongue he tries to do so, but can protrude it only just beyond his teeth. He does not complain of any pain anywhere.

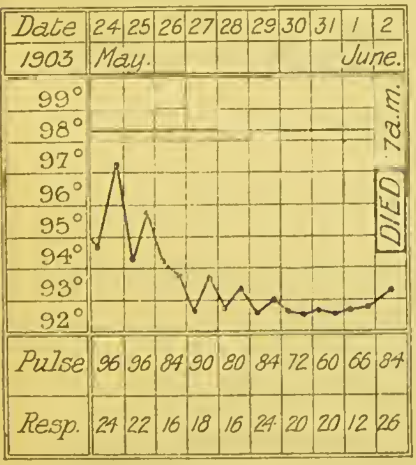
May 30. Condition worse. He lies in bed practically unconscious.

June 3. Died this morning at 7 A.M.

The following chart represents the course of the disease :—


Date 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
1903 March. April.
103° 102° 101° 100° 99° 98° 97° 96°
Pulse 100 95 90 95 96 90 90 90 96 94 90 90 72 72 84 84 84 96 90 84 96 84 100 96 96 100 84 90 84 84
Resp. 20 24 24 20 20 20 24 24 22 16 18 20 16 18 20 16 20 20 22 24 20 20 20 24 22 34 22 22 24 24


Date 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
1903 Apr. May.
104° 103° 102° 101° 100° 99° 98° 97° 96° 95° 94° 93°
Pulse 96 84 96 84 84 84 84 84 84 90 94 84 96 90 96 90 108 108 114 96 90 108 96 100 90 96 90 95 104 76
Resp. 22 24 24 22 18 20 16 24 28 20 24 20 16 18 20 20 24 24 24 20 24 24 16 20 24 24 20 24 18 20


Date 24 25 26 27 28 29 30 31 1 2
1903 May. June.
99° 98° 97° 96° 95° 94° 93° 92°
7a.m.
DIED
Pulse 96 96 84 90 80 84 72 60 66 84
Resp. 24 22 16 18 16 24 20 20 12 26

The following table shows the presence or absence of trypanosomes in the cerebro-spinal fluid and blood :—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 6.... | + | – | – | | + |
| ,, 7.... | | | – | | |
| ,, 18.... | + | | + | | |
| ,, 22.... | | – | | | |
| May 18.... | | | | | + |

June 3. *Port-mortem.*—9 hours after death.

The body is emaciated. Rigor mortis present. Abdomen retracted. Superficial lymphatic glands slightly enlarged.

*Brain.*—Weight 2 lb. 10 oz. There is considerable increase of subarachnoid fluid. Adhesion of arachnoid at one point near the middle of the median fissure. Pia mater looks like ground glass; sulci filled with fluid. On section numerous petechial hæmorrhages in the white matter of cerebrum. About 5 c.c. of fluid was obtained from the lateral ventricles. These is some congestion of the surface of the brain. Brain otherwise normal.

*Heart.*—No fluid in pericardium. Substance flabby, especially the right ventricle, and the wall is thin. No petechiæ. Valves of the right side normal. There are small atheromatous patches at the aortic orifice. Weight 8 oz.

*Lungs.*—No fluid in either pleural cavity. Slight adhesions on both sides. Left weighs 8 oz. Healthy on section. Right weighs 8 oz., also healthy on section.

*Spleen.*—Weight 1 lb. 8 oz. Substance firm, increase of fibrous tissue. Slight pigmentation.

*Liver.*—Weight 3 lb. 4 oz. Healthy on section.

*Mesenteric Glands.*—Only slightly enlarged.

*Kidneys.*—Each weigh 4 oz. Normal on section. Capsule strips off readily.

The 5 oz. of fluid from left lateral ventricle were centrifuged, and living trypanosomes were found 10 hours after death.

CASE 21.—Kidorme (Male.) Age 20. District, Bussi. Occupation, Leadworker. Food, Fish, Sweet Potatoes and Bananas.

March 24, 1903. Admitted to hospital. Patient states that his illness began with cough and pain in his chest, and that he has been ill about 3 months.

April 7. He is weak on his legs, being only able to totter along. There is trèmor of the fingers. His pupils are equal and normal. His speech is indistinct, weak and monotonous.

April 22. *General Condition.*—He is well nourished. His gait is weak and uncertain, he walks carefully with bent legs. He is intelligent, and the expression of his face is fairly bright. There is no œdema anywhere. The mucous membranes are normal. No trophic changes. Lymphatic glands are generally enlarged, about the size of beans in the inguinal region. He does not complain of any pain.

*Nervous System.*—Intelligence and memory are good. He lies about all day, but does not sleep much during the day. Speech, rather weak and indistinct. Eyes normal. Tongue jerky. Tremor of lips and hands and generally of body. His sensibility to touch is normal, as also is his muscle sense. Knee jerks are present. Ankle clonus absent.

*Alimentary System.*—His appetite is good, tongue moist, furred, and gums are healthy. The liver and spleen are not enlarged. Bowels regular, with use of medicine.

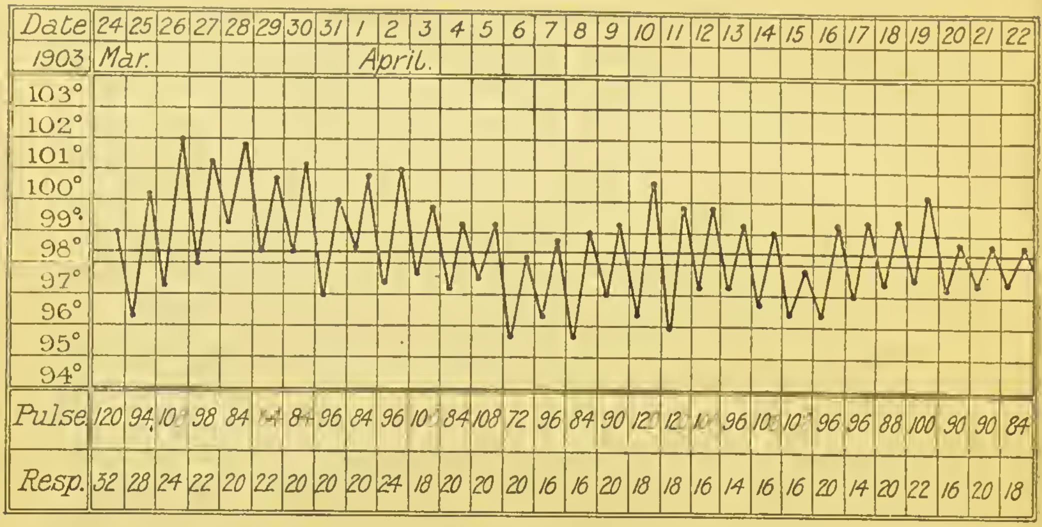
*Circulatory System.*—Apex beat is imperceptible. Heart sounds weak. No bruit. Pulse 120. Small, tension low, compressible, rhythm irregular.

*Respiratory System.*—Nothing abnormal.

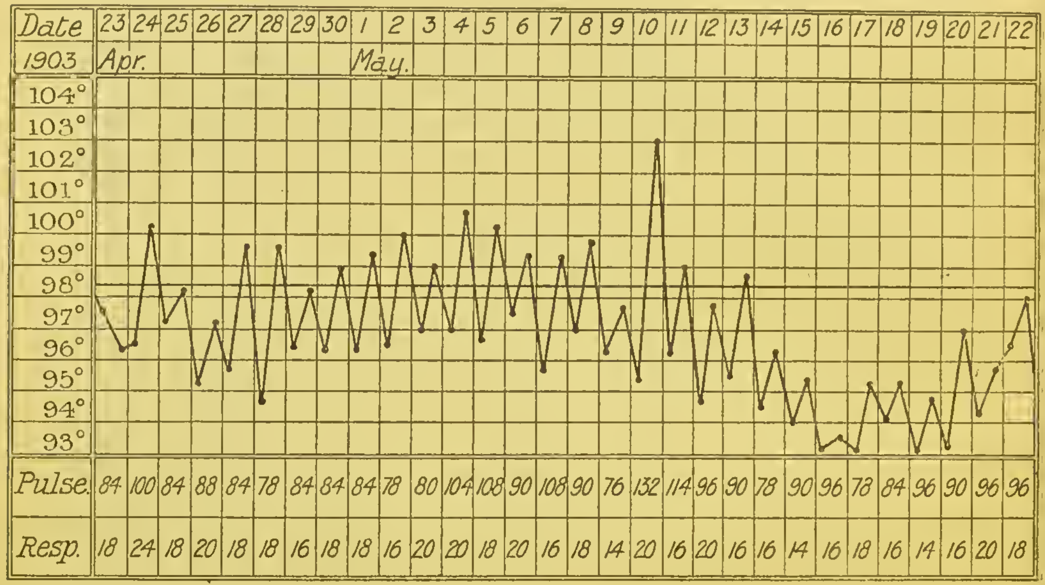
*Cutaneous System.*—Nothing abnormal.

June 10. Died 10 P.M.

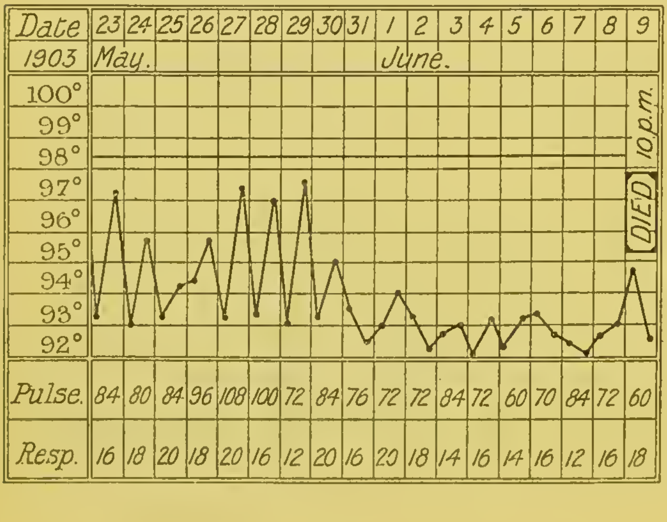
The following chart represents the course of the disease :—



| Date | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | Mar. | | | | | | | | April. | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 120 | 94 | 108 | 98 | 84 | 84 | 84 | 96 | 84 | 96 | [illegible] | 84 | 108 | 72 | 96 | 84 | 90 | [illegible] | [illegible] | [illegible] | 96 | [illegible] | [illegible] | 96 | 96 | 88 | 100 | 90 | 90 | 84 |
| Resp. | 32 | 28 | 24 | 22 | 20 | 22 | 20 | 20 | 20 | 24 | 18 | 20 | 20 | 20 | 16 | 16 | 20 | 18 | 18 | 16 | 14 | 16 | 16 | 20 | 14 | 20 | 22 | 16 | 20 | 18 |



| Date | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | Apr. | | | | | | | | May. | | | | | | | | | | | | | | | | | | | | | |
| Pulse | 84 | 100 | 84 | 88 | 84 | 78 | 84 | 84 | 84 | 78 | 80 | 104 | 108 | 90 | 108 | 90 | 76 | 132 | 114 | 96 | 90 | 78 | 90 | 96 | 78 | 84 | 96 | 90 | 96 | 96 |
| Resp. | 18 | 24 | 18 | 20 | 18 | 18 | 16 | 18 | 18 | 16 | 20 | 20 | 18 | 20 | 16 | 18 | 14 | 20 | 16 | 20 | 16 | 16 | 14 | 16 | 18 | 16 | 14 | 16 | 20 | 18 |



| Date | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | May. | | | | | | | | | June. | | | | | | | | |
| Pulse | 84 | 80 | 84 | 96 | 108 | 100 | 72 | 84 | 76 | 72 | 72 | 84 | 72 | 60 | 70 | 84 | 72 | 60 |
| Resp. | 16 | 18 | 20 | 18 | 20 | 16 | 12 | 20 | 16 | 20 | 18 | 14 | 16 | 14 | 16 | 12 | 16 | 18 |

The following table shows the presence or absence of trypanosomes in the cerebro-spinal fluid and blood :—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 28 .... | | | | | + |
| Apr. 18 .... | + | | + | | |
| „ 22 .... | | – | | | |

June 10. *Post-mortem.*—14 hours after death.

The body is that of a fairly well-nourished young man of about 20 years of age. Rigor mortis well marked. A slight superficial bed sore is seen over the lumbar region. Slight general enlargement of the lymphatic glands. Pupils equal and normal.

*Brain.*—On opening the calvarium the dura-mater appears healthy, and there are no adhesions between it and the brain.

The surface of the brain has the typical Sleeping Sickness appearance. The vessels are injected, the convolutions are flattened, and the sulci are filled with an excess of sub-arachnoid fluid, giving them a ground-glass appearance. On section the white matter shows congested points, the lateral ventricles contain about 6 c.c. of cerebro-spinal fluid, otherwise the brain appears healthy to the naked eye.

*Thorax.*—No fluid in either pleural or pericardial cavities.

*Heart.*—Weighs 8 oz. There is a slight excess of fat in the epicardium. There are no petechiæ and nothing abnormal, except a slight deposit of atheroma at the beginning of the aorta.

*Lungs.*—Left weighs 1 lb. There is some congestion of the lower lobe, otherwise the organ appears healthy. Right weighs 1 lb., and is in the same condition as the right.

*Liver.*—Weighs 3 lb. 2 oz. The surface is somewhat rough to the touch, like Morocco leather. On section the lobules stand out rather prominently, due probably to dilated peripheral capillaries. There are dark ramifying lines, evidently of fibrous tissue, enclosing several lobules, a commencing coarse cirrhosis. The tissue is bile stained, exceedingly tough, and there is probably some fatty degeneration.

*Spleen.*—The spleen weighs 8 oz. It is somewhat enlarged. It is pigmented and softened.

*Kidneys.*—Each weighs 4 oz. Capsules strip off readily. On section the tissue appears healthy.

*Alimentary System.*—The mesenteric glands are enlarged to the size of beans. The intestines appear healthy.

*Remarks.*—An ordinary uncomplicated case of Sleeping Sickness.

The accompanying photograph was taken of Kidorme a month before he died, and it must be admitted his appearance is that of an ordinary healthy native (see Plate 3).

CASE 14.—Benjameni (Male). Age 24 years. District, Bussi. Food, Fish, Bananas and Sweet Potatoes.

March 24, 1903. Admitted to hospital. Patient states that there have been no other cases of Sleeping Sickness in his house. The first symptom he noticed was that he began to sleep during the day more than usual.

April 7. This man is thin and badly nourished. His gait is ordinary. He seems intelligent, and there is nothing peculiar about the expression of his face. No œdema of any part. Lymphatic glands are enlarged generally to the size of peas, and in the inguinal and femoral regions to the size of beans.

*Nervous System.*—His speech is decidedly weak, but distinct enough. No tremors of the tongue or lips, but some slight tremor seen in the hands. Knee jerks are present. Ankle clonus absent.

*Alimentary System.*—Tongue moist and slightly furred. Lips cracked. Liver not enlarged.

*Circulatory System.*—Apex beat seen in the third space. Sounds normal. Pulse 108. Tension medium, size small, compressibility moderate, regular.

May 6. Patient is getting thinner and weaker. He seldom leaves his bed now. The tremors of hands, tongue, and body are very marked. He is passing into the third stage of the disease.

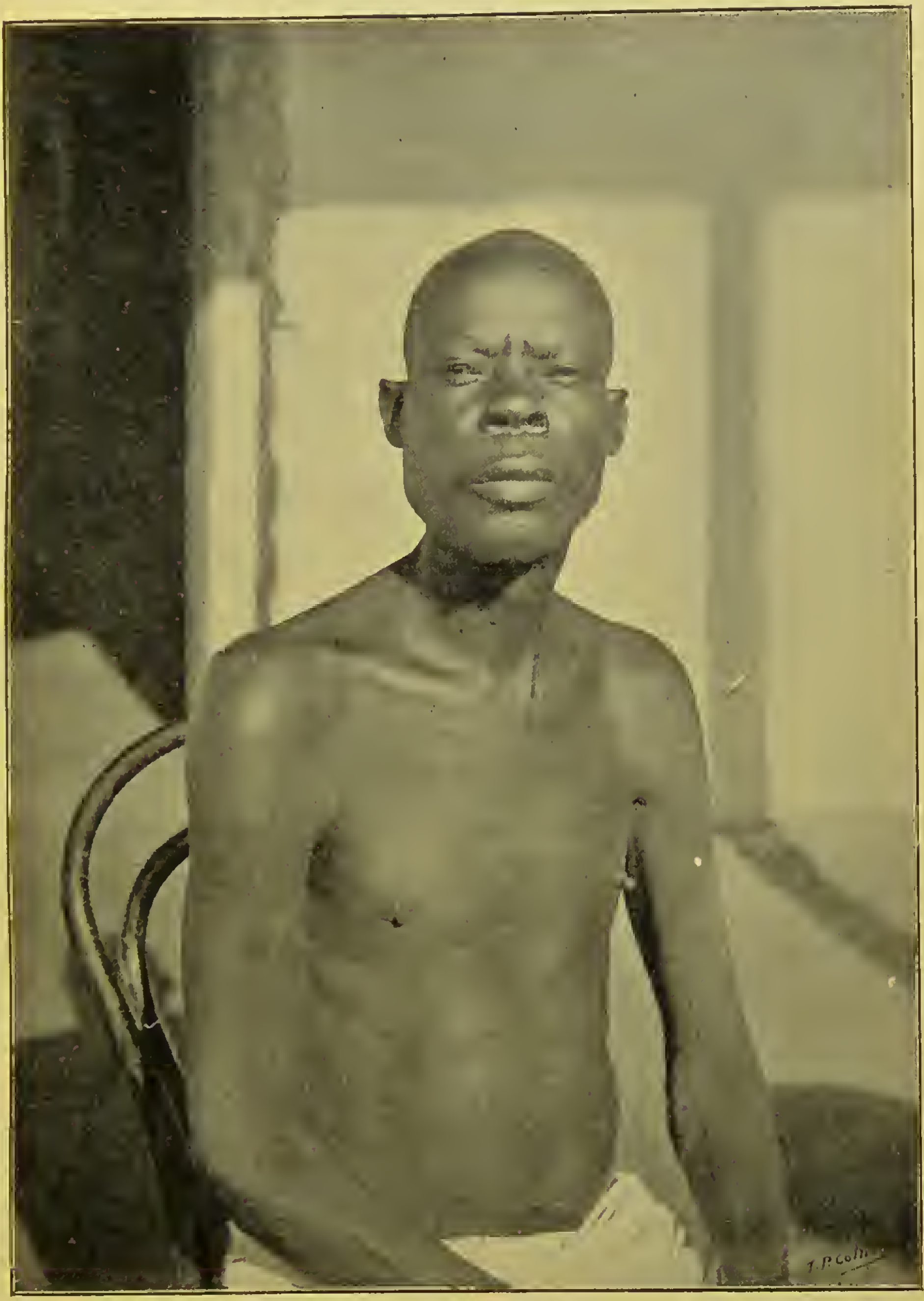
May 13. Patient is distinctly better than he was last week. He gets up every day.

May 20. Patient still gets up every day, but is very feeble.

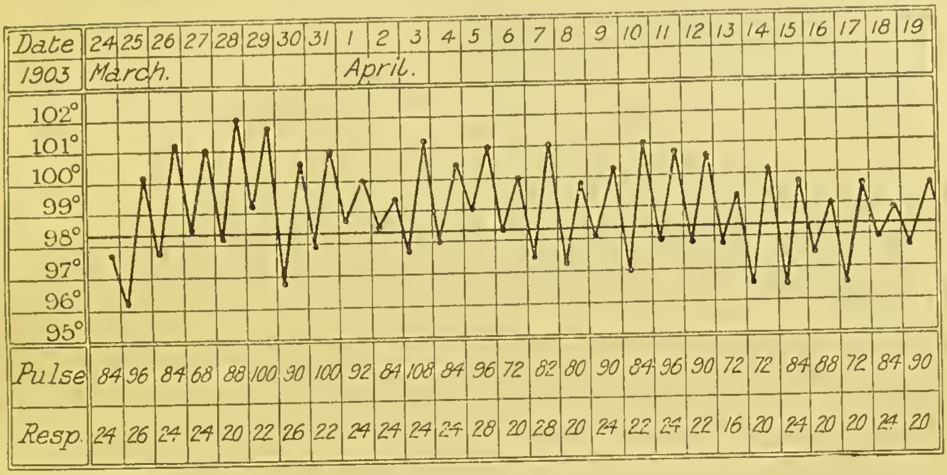
May 29. Appears worse, is very feeble, and unable to speak.

May 30. Died 11 P.M. No *post-mortem.*

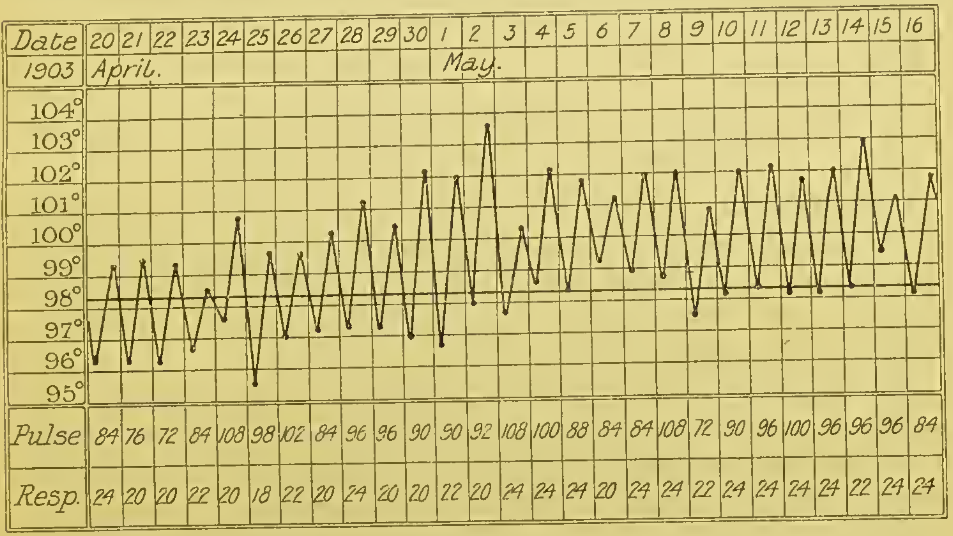
The following chart represents the course of the disease :—



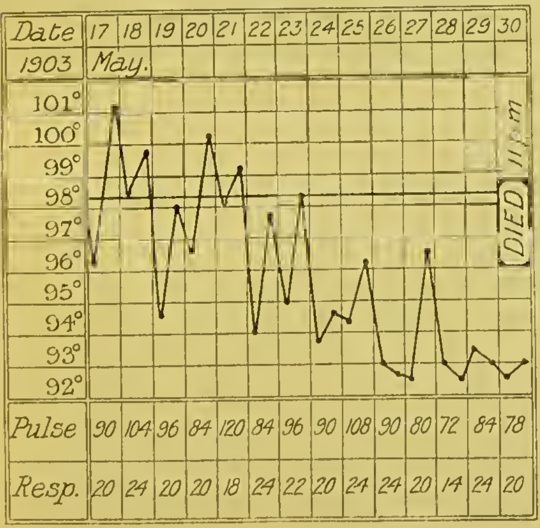
KIDORME, CASE 21.



| Date | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 31 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | March. | | | | | | | | April. | | | | | | | | | | | | | | | | | | |
| Pulse | 84 | 96 | 84 | 68 | 88 | 100 | 90 | 100 | 92 | 84 | 108 | 84 | 96 | 72 | 82 | 80 | 90 | 84 | 96 | 90 | 72 | 72 | 84 | 88 | 72 | 84 | 90 |
| Resp. | 24 | 26 | 24 | 24 | 20 | 22 | 26 | 22 | 24 | 24 | 24 | 24 | 28 | 20 | 28 | 20 | 24 | 22 | 24 | 22 | 16 | 20 | 24 | 20 | 20 | 24 | 20 |



| Date | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | April. | | | | | | | | | | | May. | | | | | | | | | | | | | | | |
| Pulse | 84 | 76 | 72 | 84 | 108 | 98 | 102 | 84 | 96 | 96 | 90 | 90 | 92 | 108 | 100 | 88 | 84 | 84 | 108 | 72 | 90 | 96 | 100 | 96 | 96 | 96 | 84 |
| Resp. | 24 | 20 | 20 | 22 | 20 | 18 | 22 | 20 | 24 | 20 | 20 | 22 | 20 | 24 | 24 | 24 | 20 | 24 | 24 | 22 | 24 | 24 | 24 | 24 | 22 | 24 | 24 |



| Date | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 | 29 | 30 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1903 | May. | | | | | | | | | | | | | |
| Pulse | 90 | 104 | 96 | 84 | 120 | 84 | 96 | 90 | 108 | 90 | 80 | 72 | 84 | 78 |
| Resp. | 20 | 24 | 20 | 20 | 18 | 24 | 22 | 20 | 24 | 24 | 20 | 14 | 24 | 20 |

The following table shows the presence or absence of trypanosomes in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Mar. 25 .... | | | | – | – |
| ,, 28 .... | | | | – | – |
| Apr. 1 .... | | | | – | – |
| ,, 7 .... | – | | – | | |
| ,, 13 .... | | | | – | + |
| ,, 16 .... | – | | + | | |
| ,, 22 .... | | – | | | |

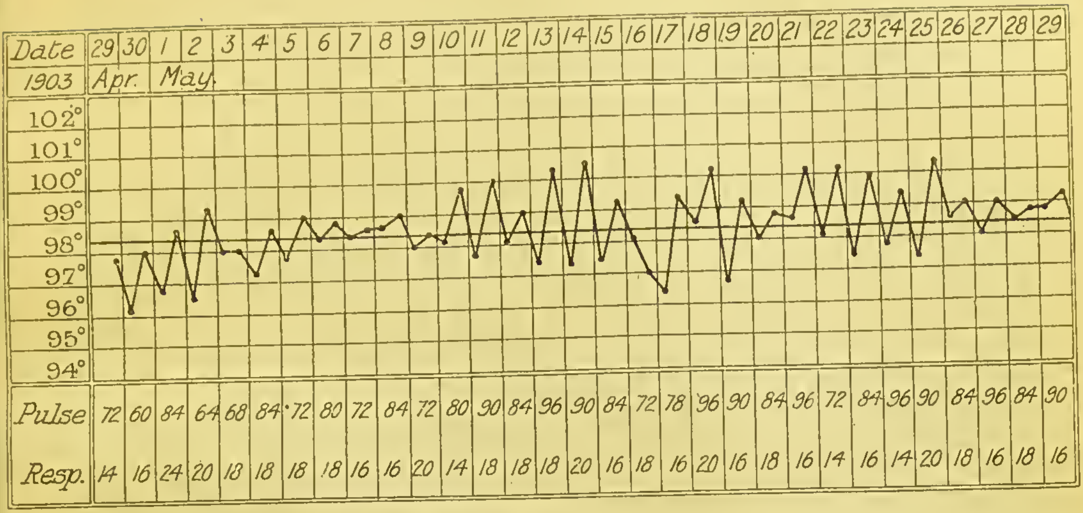
Case 79.—Festo (Male). Age 20 years. District, Kampala. Occupation, House-boy to a Missionary, for 1½ years.

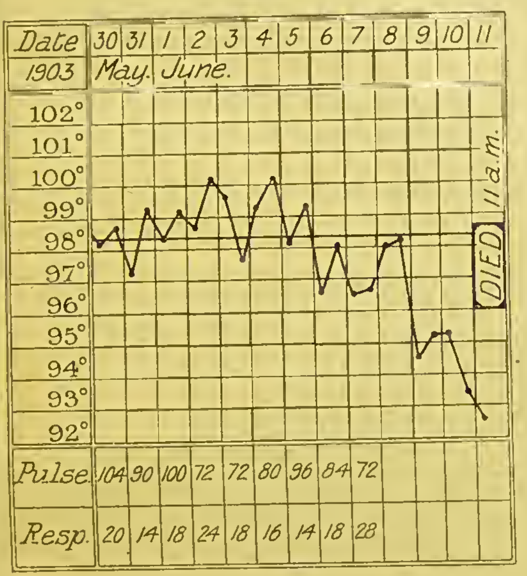
April 29, 1903. Admitted to hospital. Patient states that he has been ill about 1 month, that there had not been any other cases in the same house. A brother died of Sleeping Sickness 4 years ago at Chagwe. The first symptoms were headache and fever. Afterwards he felt drowsy during the day.

He is well nourished, but has a dull, heavy, sad expression. His speech is very weak, indistinct, and high pitched. He has tremors of tongue, lips, and slightly of hands. Pulse 120, weak, compressible. Heart. Normal no bruit. Gait weak and uncertain.

May 30. Trypanosomes *present* in cerebro-spinal fluid.

The following chart represents the course of the disease:—





CASE 44.—Nakaiba (Female). Age 10 years. District, Kampala.

April 6, 1903. This child on admission to hospital presented no perceptible signs of Sleeping Sickness, but on examining the cerebrospinal fluid, taken by lumbar puncture, trypanosomes were found.

April 29. *General Condition.*—A well-nourished child. Gait feeble, hesitating. Intelligence good. Expression not noteworthy. No œdema. Enlarged glands felt in inguinal and femoral regions, not elsewhere.

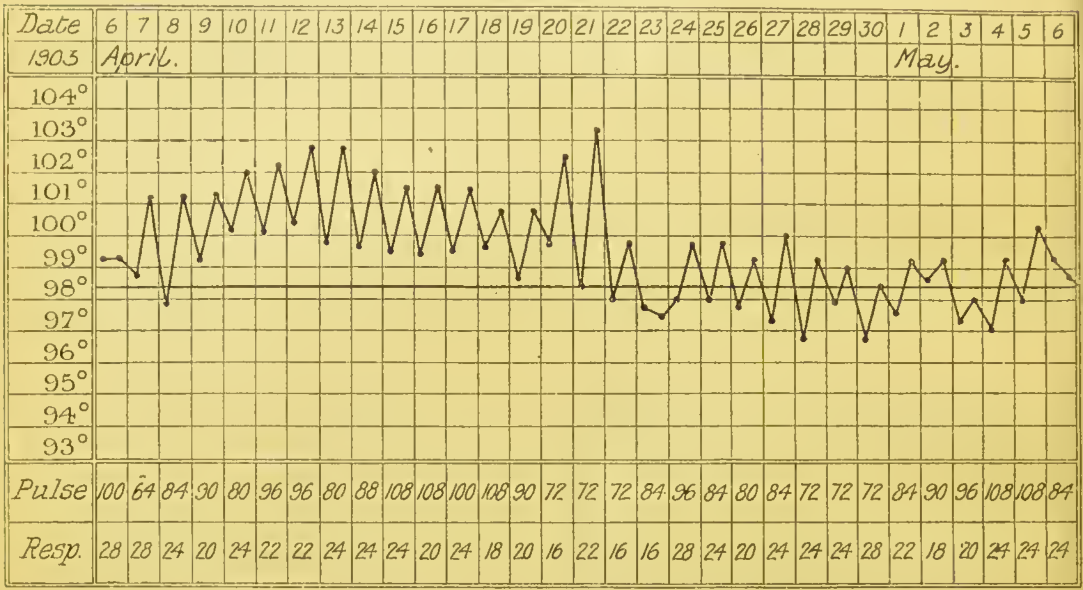
*Nervous System.*—Intelligent. Sleeps a good deal during the day. Speech, weak but distinct. No tremors of tongue, lips or hands. Knee jerks present. Ankle clonus absent.

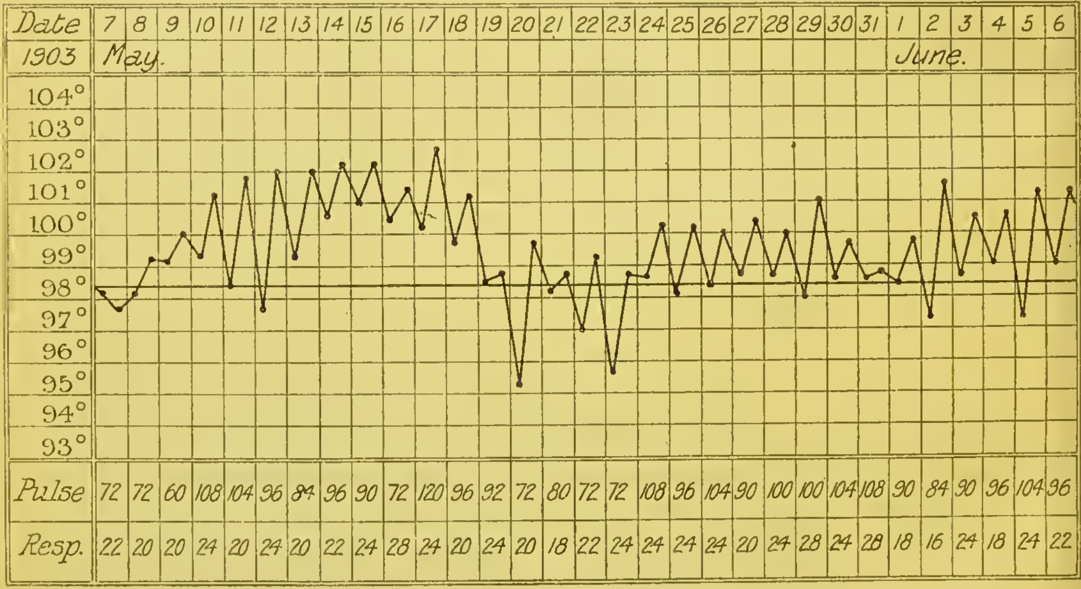
*Alimentary System.*—Tongue moist, furred. Liver and spleen not enlarged.

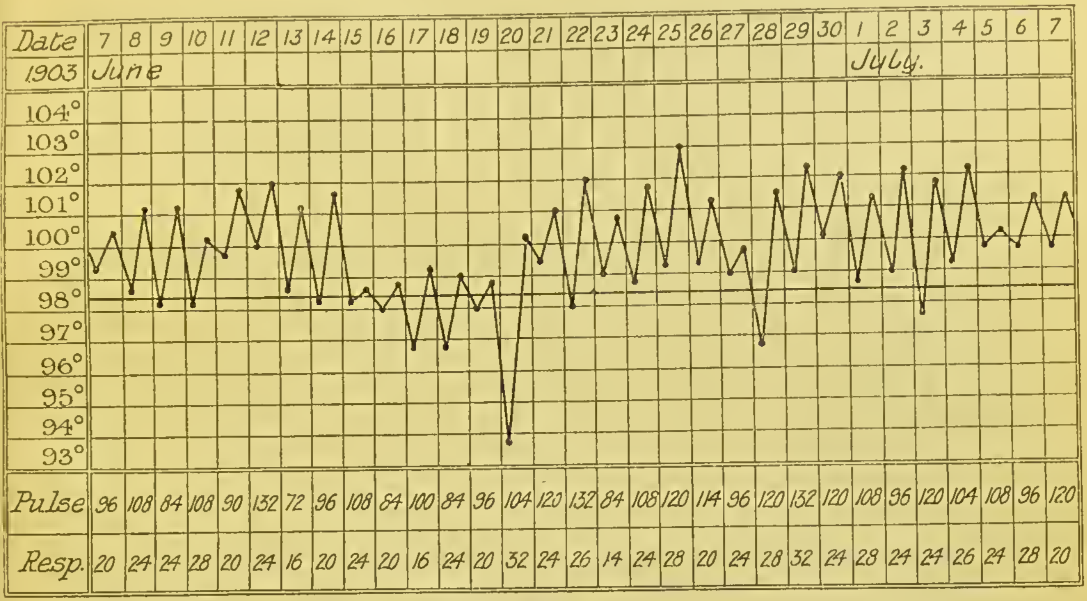
*Circulatory System.*—Heart, sounds normal. Pulse 100. Nothing noteworthy. *Cutaneous System.*—Normal. No eruptions.

July 22. Died 8 A.M. *No post-mortem.*

The following chart represents the course of the disease:—






Date
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104°
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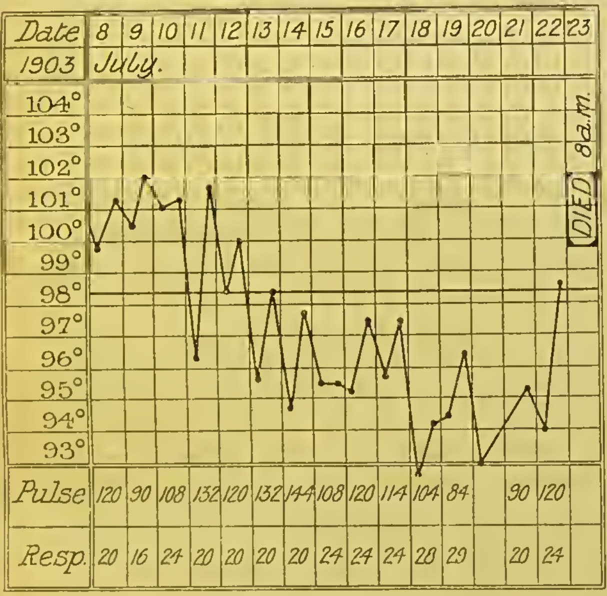
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DIED 8a.m.
Pulse
120 90 108 132 120 132 144 108 120 114 104 84 90 120
Resp.
20 16 24 20 20 20 20 24 24 24 28 29 20 24

The following table shows the presence or absence of trypanosomes in the blood and cerebro-spinal fluid:—

| Date. | Parasites in blood. | | | Parasites in cerebro-spinal fluid. | |
|---|---|---|---|---|---|
| | Filaria. | Malaria. | Trypanosoma. | Filaria. | Trypanosoma. |
| 1903. | | | | | |
| Apr. 6.... | | | | | + |
| ,, 22.... | | — | | | |
| May 12.... | | — | + | | |
| ,, 13.... | | — | | | |

*Remarks.*—This is an ordinary uncomplicated case of Sleeping Sickness of long duration. During the first months the patient remained plump and cheerful, but during the last few weeks she went down hill rapidly, became extremely emaciated and bed sores formed.

CASE 152.—Sabiri (Male). Age 16 years. District, Busoga.

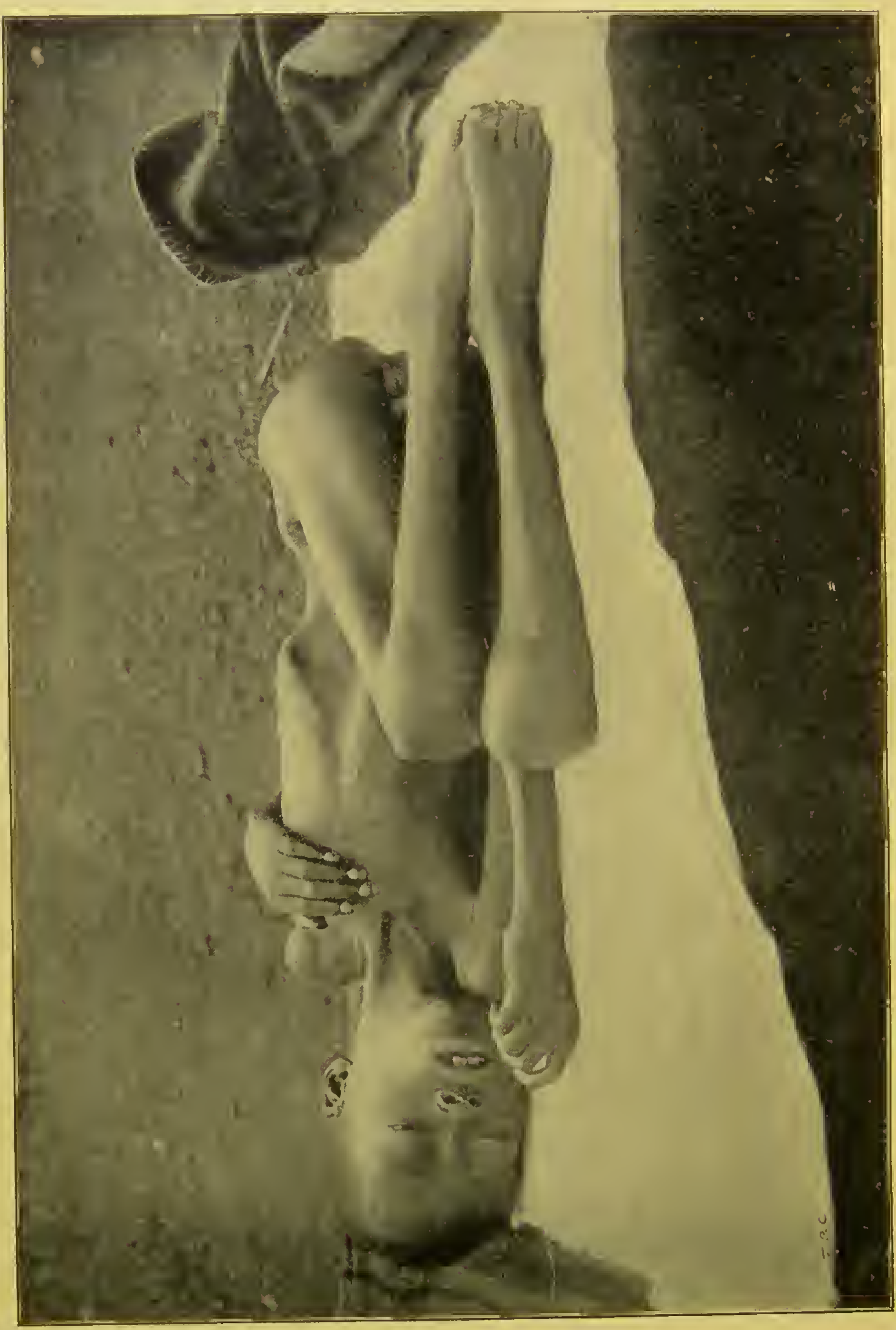
July 20, 1903. Admitted to hospital. This patient is much emaciated, he can scarcely walk. His eyes are wide open, staring. There is no tremor of the tongue or fingers. He is evidently entering the third stage.

July 27. His eyes are staring, he drinks milk out of a feeder. He can hardly walk. He complains of pains in his back and right shoulder blade. There is no headache.

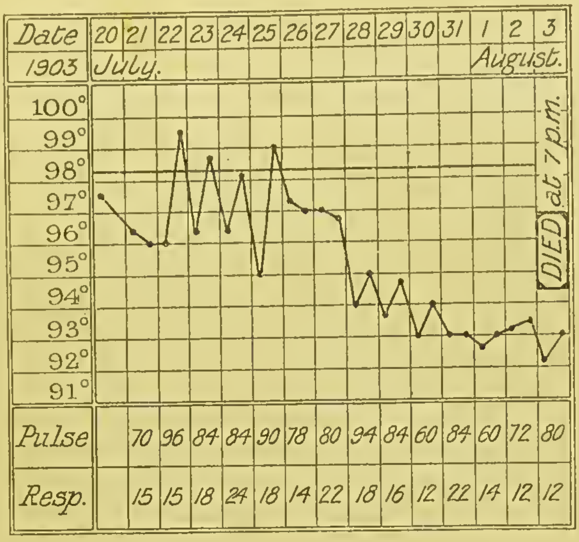
August 3. During the past week, patient has got rapidly worse. He is drowsy and lies with his eyes closed, but not asleep. This morning there is a green purulent discharge from the left nostril. There is great swelling of all the glands on the right side of the neck and jaw and retraction of the head. The eyes are to-day again widely open and patient moans constantly as if in great pain. He will not take any food now. He is in the third stage and practically moribund.

August 4. Patient died last night at 7 P.M.

The following chart represents the course of the disease:—



Sabiri, Case 152.



*Post-mortem.*—An incision was made over the swollen glands and it was found that the lymphatic glands were all intensely inflamed most being of a dark red colour. Some of the deeper cervical glands were very large and suppurating in several places.

The accompanying photograph of Sabiri was taken on August 1, and shows the patient in the last stage of the disease.

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## 9.

# A PROVISIONAL LIST OF DIPTERA, Etc.,

## Forwarded from Uganda by Lieut.-Colonel Bruce during the Investigations of the Sleeping Sickness Commission.

By ERNEST E. AUSTEN, Zoological Department, British Museum.

[Sent in November 9, 1903.]

The following list represents no more than the result of a preliminary examination of the material already received. Since collections of blood-sucking and other Diptera are still being made under the supervision of Dr. Nabarro and Captain Greig, it has been thought advisable to defer the final *working-out* of the material at present available, with the description of new species, until the conclusion of the investigations of the Sleeping Sickness Commission. Owing to the fact that the bulk of the material forwarded by Colonel Bruce was obtained by unskilled native collectors, it is unfortunately not in the best of condition, and many specimens will certainly prove to be too much damaged to determine or describe. In view of the increased importance which now attaches to Diptera as transmitters of disease, it cannot be too strongly urged that a competent Dipterist should be included in the *personnel* of any future expedition for the investigation of maladies which there may be reason to suppose are disseminated by means of insects belonging to this Order.

### Family Chironomidæ.

*Ceratopogon sp. nov.* [B]*. Mianga river: July 11, 1903.

### Family Culicidæ.

*Toxorhynchites brevipalpis*, Theob. [B]. Entebbe: in house.

### Family Simulidæ.

*Simulium sp. nov.* [B]. "Mbwa-fly," Kyagwe.

---

* The letter [B] in square brackets, following the name of a species, indicates that it is a *blood-sucking form.*

### FAMILY TABANIDÆ.*

*Chrysops stigmaticalis*, Lw. [B]. Busoga.
,, *sp. nov.* [B].
,, *sp. nov.* [B].
*Hæmatopota*—several species, all apparently new [B].
*Tabanus rubicundus*, Walk. [B].
,, *latipes*, Macq. [B]. Buvuma Island: from long grass.
,, *biguttatus*, Wied. (or closely allied species) [B]. Busoga: from swamp.
,, *fasciatus*, Fab. (or closely allied species) [B]. Bugaya Island: shore of lake.
,, *sp.*, nearly related to *T. fasciatus*, Fab. [B].
,, ? *rufipes*, Macq. [B].
,, *sp.*, nearly related to *T. pervasus*, Walk. [B].
,, ? *socius*, Walk. (*serratus*, Lw.) [B].
,, several species, as yet undetermined [B].

### FAMILY ASILIDÆ.

*Promachus fasciatus*, Fab.†

### FAMILY NEMESTRINIDÆ.

*Colax sp.*, probably new. Bulemwezi: from grass lands.

### FAMILY DOLICHOPODIDÆ.

*Psilopus carus*, Walk. Entebbe: in laboratory.

### FAMILY SYRPHIDÆ.

*Syrphus adligatus*, Wied. Chagwe.
*Asarkina ericetorum*, F.
*Eristalis tæniops*, Wied. Busoga; Jinja: from swamp.
*Helophilus*—two species, ? new. Busiro: from grass by a stream.
*Megaspis sp.*, probably new (near *Megaspis curtus*, Lw.).
*Plagiocera maculipennis*, Lw. Busoga: near river.
*Genus et sp. nov.*, allied to *Plagiocera.*
*Microdon brevicornis*, Lw. Entebbe: in laboratory.

### FAMILY MUSCIDÆ.

*Auchmeromyia luteola*, Fab. (*Ochromyia senegalensis*, Macq.). Bulemwezi.‡
*Zonochroa fasciata*, Macq. Bulemwezi and Bukoba: from long and short grass.

---

* It will be observed that no examples of the genus *Pangonia* were received.

† This species also occurs in West Africa.

‡ This species ranges from Somaliland to Natal, and is also found in West Africa. In Nyasaland it is known from the mouth of the Zambesi up to the Shire Highlands.

*Zonochroa spp.*, probably new.
*Phumosia trifaria*, Big. Bulemwezi: from grass and banana plantations.
*Glossina palpalis*, Rob.-Desv. [B].
  ,, *pallidipes*, Austen* [B].
*Pycnosoma marginale*, Wied. Bulemwezi.
  ,, *sp.*, near *P. marginale*, Wied.
  ,, *chloropyga*, Wied. Bulemwezi; Chagwe.
*Pseudopyrellia sp.*
*Lucilia sp.*
*Genus et spp.* (2) *nov.*, near *Rhynchomyia.*
*Musca sp.*, near *Musca domestica*, Linn.
*Dejeania bombylans*, Macq. (? Fab.)
*Orectocera diabolus*, Wied. Bulemwezi.
*Tachina sp.*, probably new.
*Tricholyga sp.*
*Micropalpus sp.*, ? new. Bulemwezi; Busiro: from grass by a stream.
*Exorista sp.*, Bulemwezi; Chagwe: from grass lands.
*Sarcophaga spilogaster*, Wied. Chagwe.
  ,, *sp. incert.*
*Mydœa sp. incert.* Kyadondo: from grass on lake shore.

### FAMILY ORTALIDÆ.

*Platystoma sp.*, probably new (near *Pl. punctipenne*, Walk.). Bulemwezi: from grass by a stream.

### FAMILY TRYPETIDÆ.

*Ceratitis sp.*, near *C. cosyra*, Walk. Kyadondo; Mengo: inside compound.

### FAMILY EPHYDRIDÆ.

*Dryxo sp.*, probably new.
*Genus et sp. incert.* Mitala Maryia: in papyrus swamp.

In addition to the Diptera, as enumerated above, the collection sent home by Colonel Bruce included a certain number of specimens of other Orders of insects, besides examples of two species of ticks. The names of these miscellaneous specimens are given below: they have been kindly determined by Lieut.-Colonel Bingham (Hymenoptera), Mr. W. F. Kirby (Orthoptera and Neuroptera), Mr. C. J. Gahan (Coleoptera), and Mr. R. I. Pocock (Ixodidæ).

---

* This species, together with *Glossina fusca*, Walk. [B], *Gl. longipennis*, Corti [B], and *Stomoxys tæniatus*, Big. [B], was also taken at Kibwezi, British East Africa, "in thorny bush."

## ORTHOPTERA

*Anisolabis angulifera*, Dohrn. Bulemwezi: from banana plantation.

## NEUROPTERA ODONATA.

*Palpopleura portia*, Dru.
*Ictinus ferox*, Ramb.
*Brachybasis rhomboidalis*, Beauv.

## COLEOPTERA.

*Aspidomorpha tecta*, Boh. Entebbe: in house, on flowers.

## HYMENOPTERA.

*Chalicodoma cœlocera*, Smith.
*Oxybelus arabs*, Lepel.
*Crocisa emarginata*, Lepel.
*Megachile ianthoptera*, Smith.
*Xylocopa olivacea*, Fab.
,, *sp.*, near *X. cyanescens*, Brullé.
,, *sp. incert.*
*Bembex sp.*
*Chrysis lyncea*, Fab.

## ARACHNIDA.

### ACARINA.

### FAMILY IXODIDÆ.

*Amblyomma sp.* [B], near *A. hebræum*, Koch. Entebbe: from a sheep.

*Ornithodoros moubata*,* Murray [B]. Jinja.

## APPENDIX.

Since the above lists were prepared I have received from Dr. Nabarro some additional material, including—

i. A large series of specimens of *Glossina palpalis*, Rob.-Desv. [B].
ii. Nineteen pupæ of the same species.

---

* A tube containing adults and young in formalin, accompanied by the following note:—" A bottle containing ticks named *Kibu*. They are found on the lake shore. Their bite causes inflammation with high fever, and I am told that in some cases the effect is fatal."—(W. Grant.)

iii. A series of specimens of two species of *Stomoxys** [B].

iv. A series of specimens of two species of *Musca*, one closely resembling *Musca domestica*, Linn., but distinct; the other much smaller, and possibly referable to a separate genus, since it is remarkable for the thickness of its proboscis.

The pupæ of *Glossina palpalis* are in all essential characters precisely similar in form and appearance to those of the Zululand tsetse, as figured on p. 27 of the author's "Monograph of the Tsetse-Flies" (1903). The tumid lips at the posterior extremity, however, are somewhat larger and much closer together, so that the space between them is much narrower. When this difference has once been observed, it is most noticeable, so that by means of it the pupæ of the two species are readily distinguishable. It would be interesting, from this point of view, to be able to compare the pupæ of all the species of *Glossina*, but at present material for this purpose is lacking, and these are the only species of which pupæ have so far come to hand. In size, the pupæ of *Gl. palpalis* are slightly smaller than those of the Zululand species, the specimens sent varying in length from 5¼ to 6⅓ mm.

* Neither of these is *Stomoxys tæniatus*, Big., recorded above (p. 5, note *) as having been taken at Kibwezi, British East Africa. The two species now sent, or others so closely resembling them as to be indistinguishable on a cursory examination, are already represented in the British Museum collection by specimens from Pemba Island, East Africa, August, 1899 (D. R. O'Sullivan Beare). The names of the species, which are very possibly new, must be left for future determination.

## 10.

# FURTHER OBSERVATIONS ON THE TRYPANOSOMIASES (HUMAN AND ANIMAL) IN UGANDA.

By DAVID NABARRO, M.D., and CAPT. E. D. W. GREIG, I.M.S.

(Dated Entebbe, November 20, 1903.)

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### *Introductory.*

After Colonel Bruce's departure from Uganda on August 28, 1903, we continued the investigations of the Commission on human trypanosomiasis, and studied more in detail certain animal diseases which were associated with the presence of trypanosomata. An attempt was made to ascertain whether these trypanosoma diseases in animals were identical with Nagana or Surra, or whether they were new to medical and veterinary science. Our experiments, as recorded in this Report, are not sufficiently numerous or far advanced to enable us to state definitely whether we have been studying the *Trypanosoma brucei* or *Trypanosoma evansi*, or whether any of the parasites investigated were new species (Section I).

We also continued the observations upon several Sleeping Sickness patients, and have reported a typical case occurring in a Persian which, at the time of writing the Report, we thought to be the first instance on record of the disease attacking an individual who was not a native of Africa. About the same time, although we heard of it only long afterwards, Sir P. Manson's patient (a European missionary), who had had trypanosomata in her blood for many months, died in England of Sleeping Sickness.

Amongst the patients we had under observation was a boy who suffered from recurrent attacks of fever, rather like Relapsing Fever. He was sent up as a case of Sleeping Sickness, but we could get no cerebro-spinal fluid on lumbar puncture. On examining his blood, spirochætes were found on at least two occasions, and as this was the first undoubted case of "Spirochæte" Fever occurring in that part of the world, we thought it worth putting on record. Since our case which was under observation in August, 1903, Drs. Hodges and Ross, of the Uganda Medical Service, have had other cases in native Waganda and in Indians domiciled in Uganda, which have been reported in the medical journals.

We have kept under observation and examined from time to time the five natives suffering from so-called "Trypanosoma Fever," whose cases were given in the earlier reports of the Commission. One of them, Bara Risgallah, when out in charge of a gang of prisoners one day towards the end of October, 1903, was found sound asleep on the ground and was aroused with difficulty. This was the first time that he had manifested any signs of Sleeping Sickness, if, indeed, this occurrence is to be looked upon as such at all or merely an accident.

The four other patients continue in apparently good health. Since Colonel Bruce's departure, we have failed to find trypanosomata in the cerebro-spinal fluid of J. Murjan and K. Barigi, who had previously both shown them on two occasions.

Two of our young experimental dogs developed a piroplasmosis which proved fatal in both cases. Advantage was taken of the occurrence to study the effect of injection of the infected blood from these puppies into an adult dog and a monkey. Both animals developed piroplasmata in the blood and had albuminuria. The dog died after five weeks.

We have found during our continued observations on the experimental monkeys that after some time immunity is apparently produced, and that a repeated injection of the same trypanosoma (either the blood or the cerebro-spinal human trypanosoma) fails to produce the reappearance of the parasite in the peripheral blood two or more weeks after the injection.

We have arranged to inject these "immunised" monkeys with the other variety of trypanosoma to see whether *it* will develop in their blood, for if it do not, it will tend to prove that the trypanosomata occurring in the blood in "Trypanosoma Fever" and in the cerebro-spinal fluid in Sleeping Sickness are identical (Section II).

We also endeavoured to ascertain whether there is a specific carrier for each variety of trypanosoma. Our experiments seem to negative this idea, for we found that the *Glossina palpalis* was able to convey, by feeding, at least two of the animal trypanosomata from infected to healthy monkeys. Experiments with other biting flies, such as Stomoxys and Tabanidæ, yielded negative results, the former apparently being unable to convey trypanosomata to healthy animals by feeding, while the latter refused to feed when in captivity and soon died off.

Dissections of flies (Glossinæ and Stomoxys) at varying intervals after a feed of blood containing trypanosomata, yielded interesting results. It was found that the trypanosoma of Sleeping Sickness was motile in the stomach contents of *Glossina palpalis* 71 hours after a feed. The various animal trypanosomata were found active 100 hours, 5½ hours and 20 hours respectively in the stomach contents of Glossinæ, whereas the same three animal parasites were found active in the stomach contents of Stomoxys 24 hours, 12 hours and 30 hours respectively after a feed (Section III).

The very important question whether the trypanosoma of Sleeping

Sickness can be conveyed by the tsetse flies met with in the East Africa Protectorate must apparently be answered in the positive, as the result of our experiments. Owing to the importance of this question and to the possibly far-reaching consequences of the importation of infected natives from Uganda into an area where the carriers of infection exist, these experiments are to be, or already have been, repeated (Section IV).

Finally, we have given some additional data concerning the distribution of tsetse flies in Uganda, notably in the region of the Albert Nyanza, the whole of which lake appears to be surrounded by a "fly" area (Section V).

## Section I.—TRYPANOSOMATA FOUND IN THE LOWER ANIMALS IN UGANDA AND EAST AFRICA.

While the Sleeping Sickness Commission was carrying on its investigations on the human trypanosoma, it was discovered that several species of lower animals suffered from trypanosoma infections. Experiments were therefore carried out to ascertain whether these animal trypanosomata bore any relation to the trypanosomata found in the human subject.

For the purpose of reference we have numbered the trypanosomata T. I, T. II, etc., until their identity or otherwise has been established:—

T. I is the trypanosoma found in Sleeping Sickness in the cerebro-spinal fluid.

T. II is the trypanosoma found in the blood of the patients Jardien Murjan, Karala Barigi, Kamsar Sabba, Bara Risgallah, and Tabula. (See Earlier Reports of the Commission.)

T. III is the trypanosoma found in the blood of sick transport cattle (oxen) in Entebbe.

T. IV is the trypanosoma found in the blood of a large herd of sick cattle at Jinja, Busoga.

T. V is the trypanosoma found in the blood of a dog which was sent up to us from East Africa.

T. VI is the trypanosoma found in the blood of a sick mule in Entebbe.

### (a) *A Trypanosoma Occurring in Diseased Oxen in Entebbe, Uganda.* (T. III.)

On June 3, 1903, three government oxen were examined because they were out of condition. Trypanosomata were found in blood films of one (Experiment 152), but not in films of the other two.* Of the latter, one showed trypanosomata on centrifuging 10 c.c. of blood, while the other did not.

---

* See Reports of the Sleeping Sickness Commission, No. IV, pp. 47 and 48.

On inquiry it was ascertained that these oxen came from the East Africa Protectorate towards the end of 1900, passing through Busoga about September, 1900. They kept remarkably well in Entebbe until they were placed near the forest.

On August 13, the ox which had previously shown trypanosomata in films, was again brought up to the laboratory, this time time nothing more than a hide-bound skeleton. No trypanosomata were seen in a cover-glass preparation, but three days later, many were found on centrifuging 10 c.c. blood. A dog which had been injected with some of the blood on June 3, and which failed to show the trypanosoma in its blood, was reinjected on August 16, and again failed to develop trypanosomata in its blood. The ox went slowly down hill and died on September 28, and in spite of several examinations of 10 c.c. of blood, no trypanosomata were seen after August 16.

On September 5 the superficial lymphatic glands were greatly enlarged, and the animal was seen by a local trader, who said the disease was known locally as "Mukebi." While Colonel Bruce was in Entebbe, we had examined several animals in the last stages of this disease, and failed to find trypanosomata in 10 c.c. of blood.

On September 8, six other transport oxen were examined, but no trypanosomata were found in any of the blood films. The animals examined were all thought to be healthy.

On October 14, blood films of two sick oxen were examined, but no trypanosomata found.

The interesting points about this trypanosoma are (1) that it became very scanty in the blood of the sick ox towards the end of life, and could not be demonstrated during the last six weeks even by the centrifuge; (2) that the trypanosoma did not appear in the blood of a dog injected with the ox blood, although so numerous in the latter as to be seen in cover glass preparations.

### EXPERIMENT 152. Transport Ox, Entebbe. (T. III.)*

Came from East Africa about end of 1900.

June 3, 1903. Animal seedy and brought to the laboratory. Trypanosomata found in cover glass preparations. Dog 128 injected. Animal returned to work.

August 13. Animal again brought up to the laboratory, a "hide-bound skeleton." No trypanosomata found in cover glass preparations.

August 16. Trypanosomata found in numbers on centrifuging. Injected dog 128 again.

August 19. Trypanosomata absent from films.

September 5. Animal is getting gradually thinner. The superficial lymphatic glands are greatly enlarged, and the ox looks like a typical case of "Mukebi." About 30 c.c. blood centrifuged, but no trypanosomata found. Lumbar puncture was performed, but only blood obtained.

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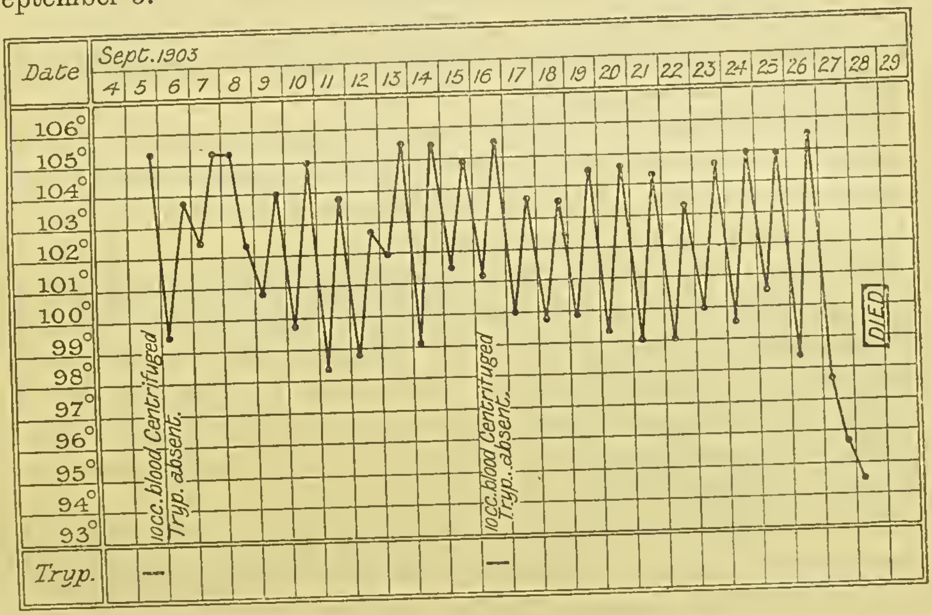
* See Reports of the Sleeping Sickness Commission, No. IV, p. 48 and Plate 2.

September 16. Blood again examined by centrifuge, but no trypanosomata found.

September 28. The ox has been gradually getting weaker, but until yesterday was able to walk about and graze with the other cattle. This morning his temperature fell to 94°·6 F., and he was unable to walk. He died at 11.30 A.M. Some blood was taken at death and injected into a dog, 207, and a monkey, 206.

The following chart represents the temperature curve from September 5 till death. No temperature observations were made before September 5.



*Post-mortem* immediately after death. Animal emaciated. Superficial lymphatic glands enlarged and congested on section. A little jelly-like material present in the subcutaneous tissues. On opening the body, a little clear fluid escaped from the pericardial cavity; none from the pleural or peritoneal cavities. The heart was pale; had yellow jelly-like material at the base, and on section two small petechiæ in the left ventricle. The lungs were healthy. The liver contained two flukes, but was otherwise normal. The gall bladder was distended with bile. The spleen was slightly enlarged. The kidneys were pale, but otherwise healthy. The retroperitoneal and mesenteric glands were only slightly enlarged. There was opacity of the left cornea, none of right.

*Remarks.*—Two injections of blood from this ox into a dog failed to infect the dog, although it received many trypanosomata on each occasion. The disease is therefore not Nagana in all probability, but its trypanosoma may be closely allied to, if not identical with, that of human trypanosomiasis. The injection of blood, taken from the ox *post-mortem*, into a dog and a monkey yielded negative results, but it must be remembered that centrifuging 10 c.c. or more of the ox blood failed to show the presence of trypanosomata at any time during the last six weeks of the animal's existence.

### (b) *A Trypanosoma Occurring in Diseased Cattle at Jinja, Busoga* (T. IV).

These cattle were brought in from the Bukedi country about May, 1903. They came from Wamia's district, to the south-west of Mount Elgon. On the way from Bukedi to Busoga they halted at Igagas, Kibue, Baleale, and Kitindis, in all of which places a tsetse fly (*Glossina pallidipes*) is found. When first brought into Jinja the cattle were apparently healthy, but later on died at the rate of five or six a day. The duration of the disease, as gathered from local information, is possibly two to three months.

*Symptoms.*—Animals are thin but not markedly emaciated. Anæmia is marked. Some slight enlargement of the lymphatic glands. The animals die often quite suddenly and in fairly good condition. Sometimes they waste, dying for several days.

*Post-mortem.*—The cervical glands are slightly enlarged and congested, sometimes hæmorrhagic. The supra-clavicular glands are also enlarged and congested. In some cases there is an increase of fluid in the pericardial and pleural cavities. The heart has yellow jelly-like material at the base and often petechiæ on its external and internal surfaces. Lungs are normal. The urine is clear. Kidneys and liver healthy. The spleen is slightly enlarged.

The native name of the disease is "Sutoko" and it has been thought to be an internal form of "Mukebi." When only the external glands are enlarged, many cases are said to recover. We examined several cases of so-called external "Mukebi" and were unable to find trypanosomata in any of the blood films taken. The native treatment is the application of a blister to the enlarged glands in the form of the juice of a plant called "Nandera," a Euphorbia found in Busoga and Uganda. The animals are said to recover after this treatment in many cases.

The following table (p. 15) gives the details of the cattle whose blood was examined for parasites.

At Jinja the total number of Bukedi cattle was 210; the blood of 13 of them was examined by films, and the trypanosoma found in 8 = 61·5 per cent. Within one month of the blood examination, 50 out of the 210 had died, probably all from this trypanosoma disease = 24 per cent.

At Kitindis, the total number of Bukedi cattle was 102. The blood of 15 of them was examined by films and the trypanosoma found in

Table I.

| No. | Date. | Place. | Animal. | Tryp. in films. | Remarks. |
|---|---|---|---|---|---|
| 1 | 10.8.03 | Jinja | Cow | Present, numerous | |
| 2 | 10.8.03 | ,, | ,, | ,, | Died on Aug. 17. Dog 153 injected on 12.8.03 |
| 3 | 11.8.03 | ,, | Young bull | Absent | Typical case of Mukebi (Grant) |
| 4 | 11.8.03 | ,, | Bull, dying | Present | *Post-mortem.* Typical |
| 5 | 11.8.03 | ,, | Bull | Absent | Lying down — ? sick |
| 6 | 11.8.03 | ,, | ,, | Present | Sick |
| 7 | 11.8.03 | ,, | ,, | ,, | ,, |
| 8 | 11.8.03 | ,, | ,, | Absent | |
| 9 | 11.8.03 | ,, | Calf | ,, | ,, |
| 10 | 11.8.03 | ,, | Bull | Present | |
| 11 | 11.8.03 | ,, | ,, | ,, | |
| 12 | 11.8.03 | ,, | ,, | ,, | |
| 13 | 11.8.03 | ,, | Calf | Absent | |
| 14 | 12.8.03 | Kitindis | Bull | Present | |
| 15 | 12.8.03 | ,, | ,, | Absent | |
| 16 | 12.8.03 | ,, | ,, | ,, | |
| 17 | 12.8.03 | ,, | ,, | Present | |
| 18 | 12.8.03 | ,, | ,, | Absent | |
| 19 | 12.8.03 | ,, | Young bull | Present | Sick. Animal killed. *Post-mortem.* Lymph. glands of neck enlarged and hæmorrhagic; also iliac glands. Heart shows no jelly, but a few small petechiæ under endocardium of L. ventricle. Blood very watery. Spleen slightly enlarged |
| 20 | 12.8.03 | ,, | Bull | Absent | Typical case of Mukebi |
| 21 | 12.8.03 | ,, | ,, | ,, | |
| 22 | 12.8.03 | ,, | ,, | ,, | Said to have recovered from Mukebi |
| 23 | 12.8.03 | ,, | ,, | ,, | |
| 24 | 12.8.03 | ,, | ,, | ,, | |
| 25 | 12.8.03 | ,, | ,, | ,, | |
| 26 | 12.8.03 | ,, | ,, | ,, | |
| 27 | 12.8.03 | ,, | ,, | ,, | |
| 28 | 12.8.03 | ,, | ,, | ,, | |
| 29 | 17.8.03 | Entebbe | ,, | ,, | The after history of these animals is uncertain |
| 30 | 17.8.03 | ,, | ,, | ,, | |
| 31 | 17.8.03 | ,, | ,, | ,, | |
| 32 | 17.8.03 | ,, | ,, | Present | Sick. Died on 20.8.03 |
| 33 | 17.8.03 | ,, | ,, | Absent | After history is uncertain |
| 34 | 17.8.03 | ,, | ,, | ,, | |

3 = 20 per cent. Within one month of the blood examination, six of these had died = 6 per cent.

The animals 29—34 were examined in Entebbe but came from the Bukedi district originally, *viâ* Jinja.

Mr. Grant, the Sub-commissioner of Busoga, considered the herd at

Kitindis to be healthier than that at Jinja, and our examination would seem to bear out this statement. Mr. Grant kept a private herd of about 20 head of cattle at Jinja, and these animals are all healthy up to the present.

On November 12 Mr. Grant wrote to inform us that between September 11 and November 12, 24 more bulls and 7 bull calves had died at Jinja and 4 bulls and 2 bull calves at Kitindis.

Within three months of the blood examination, therefore, 81 out of the 210 Bukedi cattle had died at Jinja = 39 per cent., probably all from the trypanosoma disease. At Kitindis, 12 out of 102 had died within three months = 12 per cent.

We were unable to study the complete course of the disease as it occurred naturally in cattle, so we injected into an ox 10 c.c. blood containing the "Jinja" trypanosoma. The parasite had, however, been passed successively through a monkey (Experiment 135) and a dog (Experiment 164) after being obtained from a sick ox (No. 32 in Table I). The parasite appeared in the peripheral blood films of the ox (162) 12 days after injection.

The details of this experiment down to November 17 are as follows:—

### EXPERIMENT 162.—Ox.

To note the effect of the subcutaneous injection of blood containing the "Jinja" trypanosoma (T. IV).

September 9, 1903. Injected 10 c.c. blood subcutaneously, from dog (164) containing T. IV.

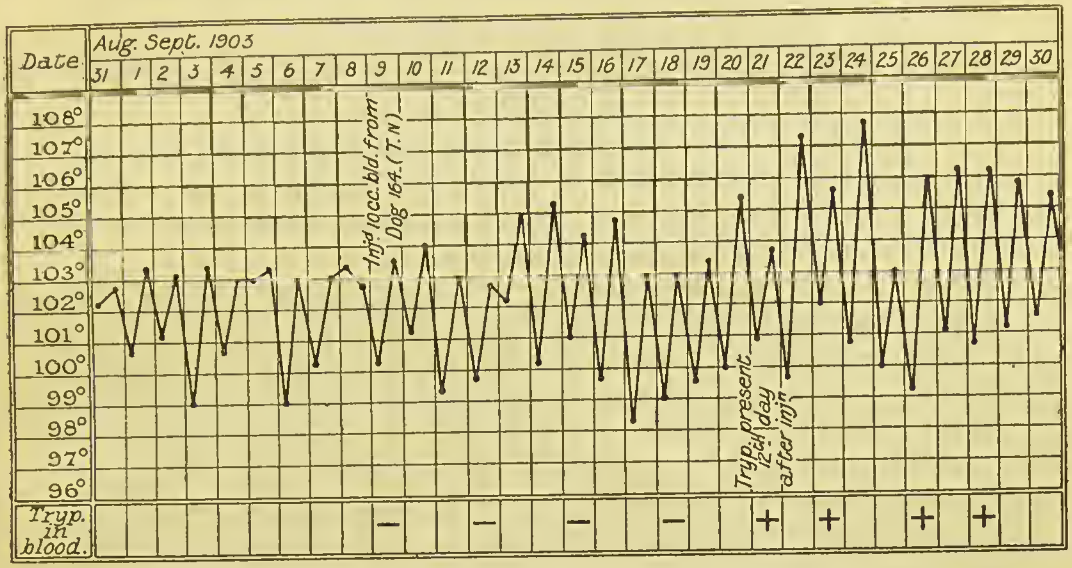
September 21. The ox has been looking thinner and rather seedy from a few days. Trypanosomata were found in blood films to-day—12 days after injection. They are thick and have very blunt posterior extremities. The flagella are very short. In these respects they resemble the original trypanosomata seen in the Jinja cattle (see Plate 1, *c*).

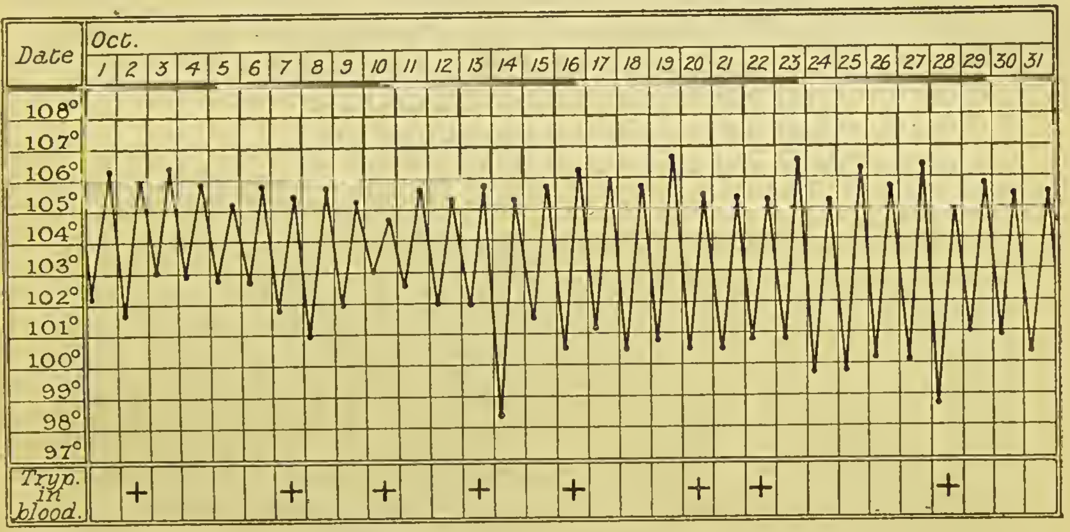
October 8. To-day the ox was noticed to lie down a good deal while out grazing with the other cattle. There is slight opacity of both corneæ.

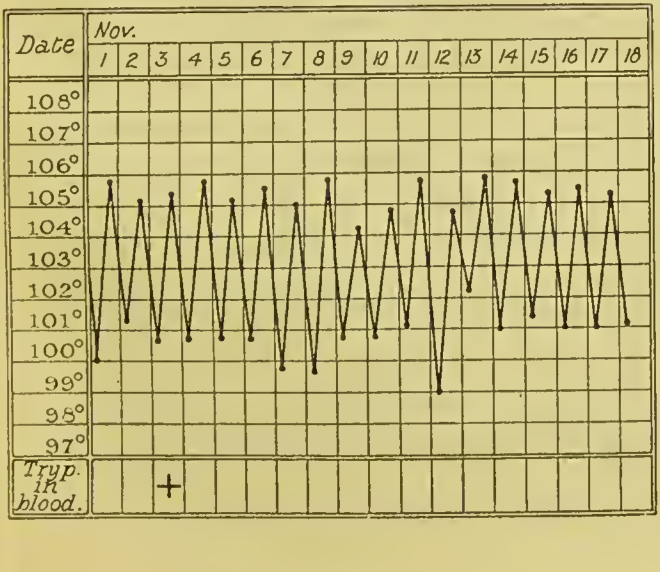
October 28. The animal is in better condition again and does not lie about when grazing. The opacity of the corneæ has not increased. The coat is in good condition.

The following chart (p. 17) gives the temperature curve and the presence or absence of trypanosomata in blood films.

November 17. This animal still shows the trypanosoma in its blood and continues in fairly good condition.


Date
Aug. Sept. 1903
31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
108° 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
Injd 10cc. bld. from Dog 164. (T.N.)
Tryp. present 12th day after injn
Tryp. in blood.
− − − − + + + +


Date
Oct.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
108° 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
Tryp. in blood.
+ + + + + + + +


Date
Nov.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18
108° 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°
Tryp. in blood.
+

## *The "Jinja" Trypanosoma* (T. IV). *Its Morphology and Pathogenicity for Animals.*

In the blood of naturally infected cattle, this trypanosoma was often very short and stumpy, with a very short flagellum and a blunt posterior end. This, however, was by no means always the case, as the following details will show. (See Plate 1.)

Case 1.—Trypanosomata very scanty; average length 24 μ, long and slender; flagellum about 8 μ.

Case 2.—Tryp. very numerous and of various sizes. The majority were short and stumpy, about 13 μ long, the flagellum being only 2—3 μ.

Others were 20—25 μ long, the flagellum about 8—10 μ. One very large parasite was seen in this film 44 μ long, with flagellum about 18 μ. It had a very pointed posterior extremity, and single nucleus. The centrosome had just divided. (See Plate 1, *b*.)

Case 3.—Tryp. very numerous. Average length 12 μ; one was only 7·5 μ long.

Case 4.—Tryp. very scanty; average length 22 μ.

Case 5.—Tryp. very scanty; average length 13 μ.

Case 6.—Tryp. numerous; average length 12—13 μ; one seen only 7 μ long.

Case 7.—Tryp. scanty; average length 17—18 μ.

Variations in size and form were also seen in the blood of inoculated animals. In Ox 162, experimentally inoculated from a dog, in Dog 164 and in Goat 192, the trypanosomata were all short and stumpy, with a very short flagellum. In Monkeys 135 and 154, and in the guinea pig (185) the parasites were long and thin and had a pointed posterior extremity; whereas in a sheep (193) on several occasions the trypanosomata were mostly very small, and some showed vacuolation and deformity of shape. This vacuolation and deformity of shape were noted also on one occasion (September 12) in Monkey 135.

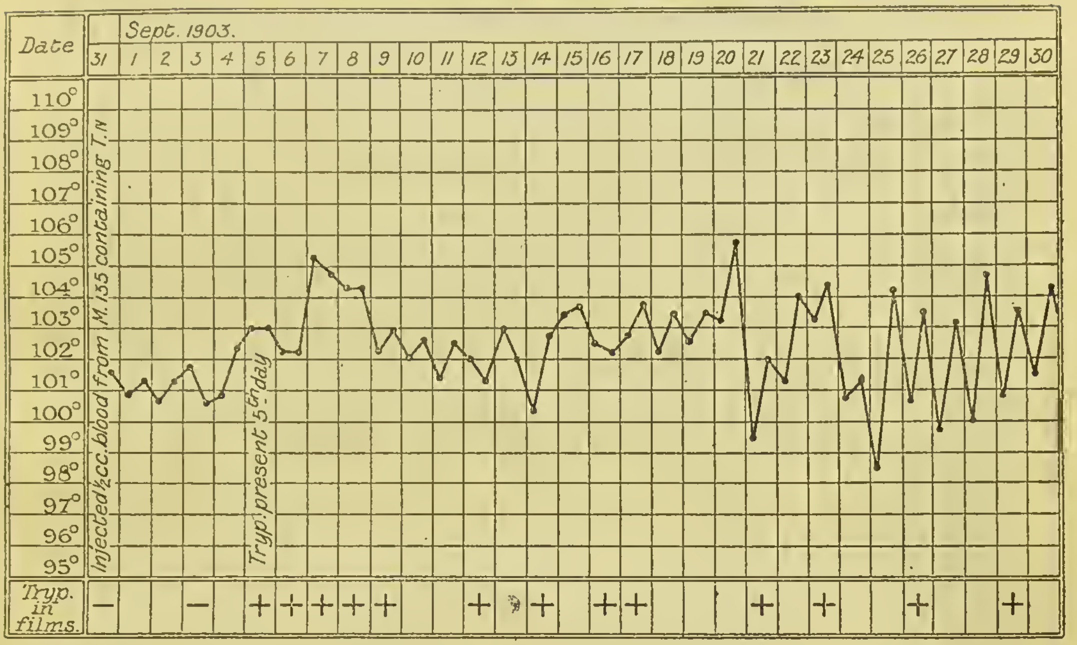
The following were the effects noted after inoculation into animals:—

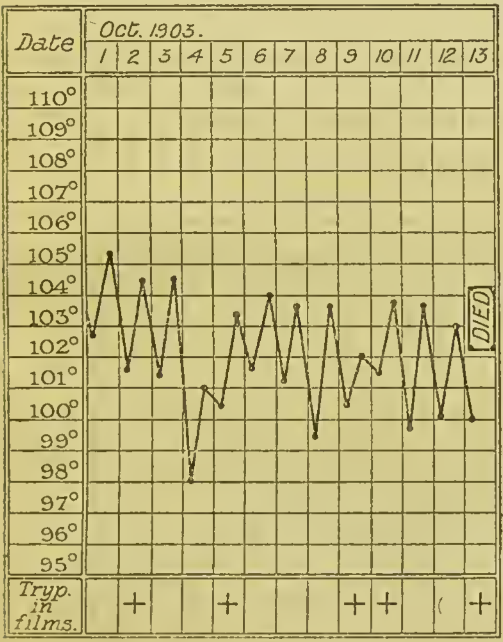
*Ox.*—Trypanosomata were seen in films of the peripheral blood 12 days after injection. This experiment is given in detail on p. 16.

*Dog.*—Trypanosomata appeared in blood films in from 5 to 10 days after inoculation. The animals succumbed in 11 days (two), 26, 29, and 43 days after injection.

The following chart shows the course of the temperature in a typical case of infection in the dog:—

### EXPERIMENT 164.—Dog (Native).



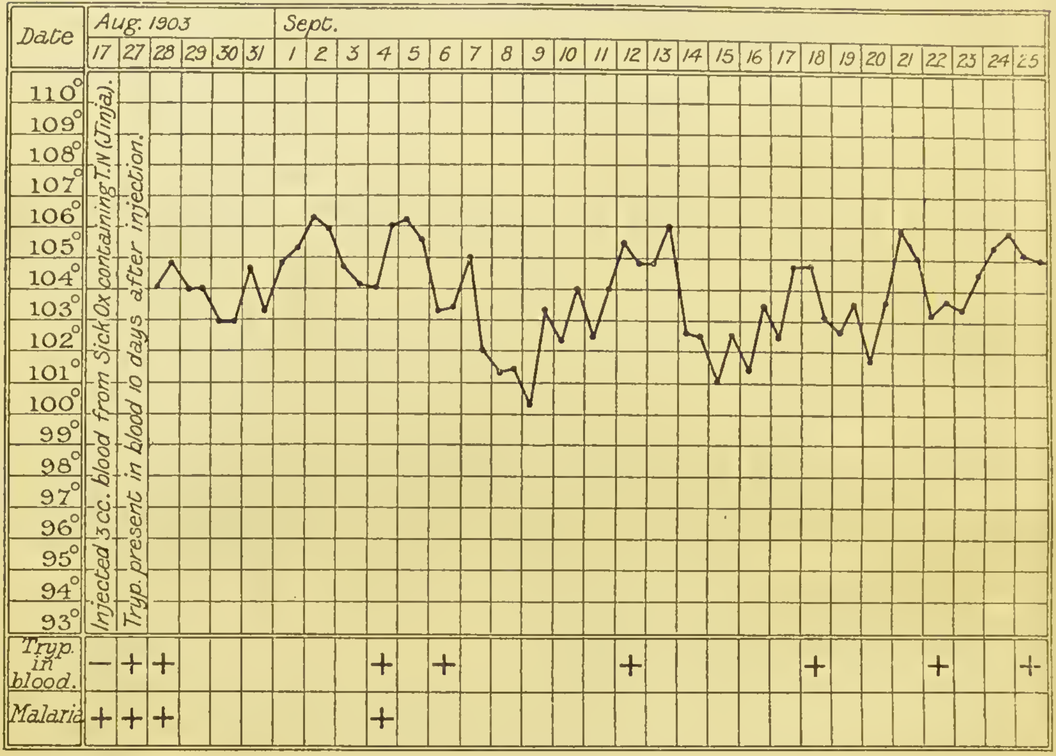


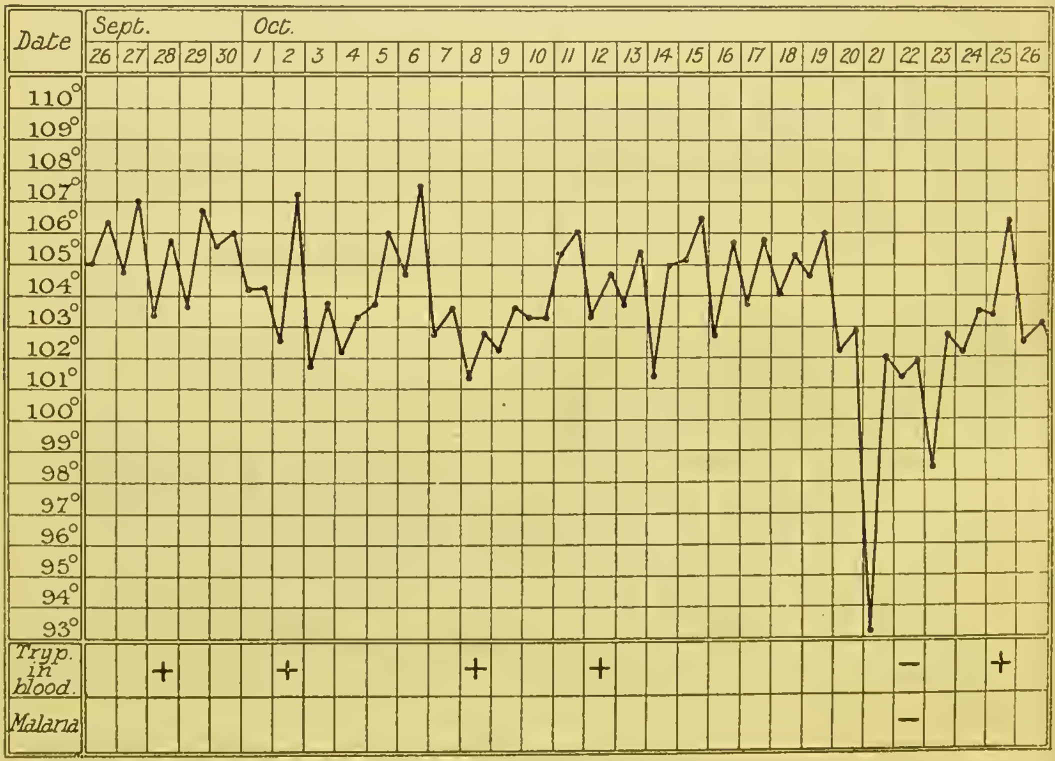
*Post-mortem*, the spleen was found markedly enlarged in all cases. In most of the animals the lymphatic glands in the neck and abdomen were enlarged and congested, sometimes hæmorrhagic.

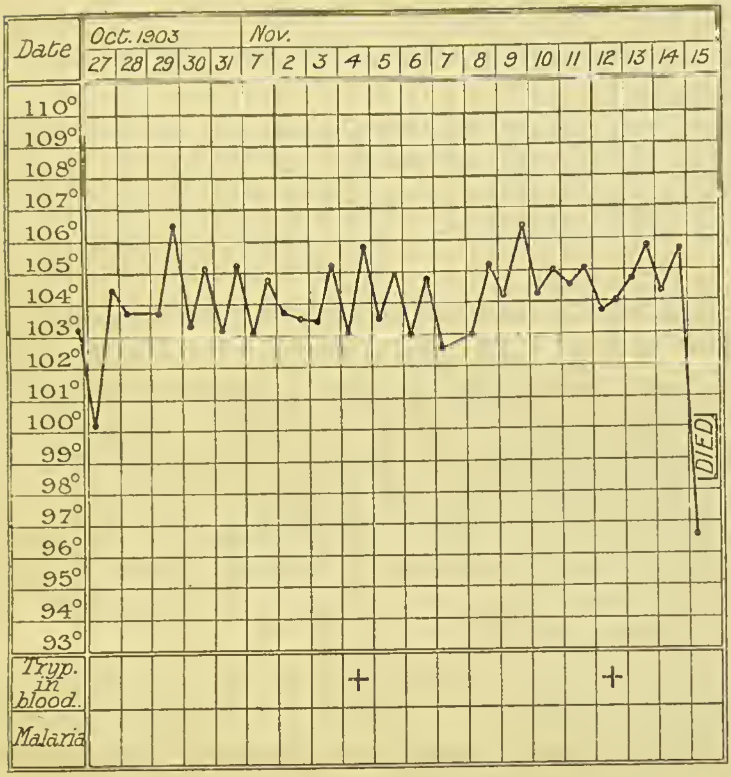
Four of the five dogs had petechiæ under the pericardium or endocardium. Two had embolic areas in the lungs, and one dog (164), which lived 43 days after inoculation, had slight opacity of one cornea.

*Monkey.*—In two experiments trypanosomata appeared in the blood 10 and 11 days after injection. One animal (135) died on the 39th day

EXPERIMENT 154.—Monkey (*Cercopithecus* sp.).







from traumatic (?) septic peritonitis, the other (154) died 90 days after inoculation. The preceding chart (pp. 20 and 21) shows the temperature curve and the presence or absence of trypanosomata in the blood.

The autopsy was rather interesting, so we append the notes in full.

*Post-mortem* immediately after death. Body rather thin. No enlarged glands or opacity of corneæ. No increase of pericardial or pleural fluid. The heart showed marked petechiæ all over the surface and jelly-like material round the base. There were signs of endocarditis, and a small ulcer was present on the auricular surface of the mitral valve. Vegetations were present in the left auricle. There were numerous petechiæ under the endocardium of both ventricles.

The lungs showed several small embolic areas.

Peritoneal fluid was not increased. The spleen was distinctly enlarged; on section its substance was firm and dark in colour.

The liver was normal. Both kidneys were studded with small hæmorrhages, on the surface and in the substance. They resembled the embolic areas seen in the lungs. There were no enlarged glands in the abdomen. Smears of the lungs, spleen and kidney showed nothing noteworthy.

In both these monkeys the disease was rather of the chronic variety, and *post-mortem* each showed signs of septic infection. This seems to indicate that in chronic trypanosoma infections, the resisting power of the individual may be so diminished as to render him more susceptible to bacterial invasion. The terminal infection with streptococci, pneumococci, etc., so commonly seen in Sleeping Sickness, is probably to be similarly explained.

Another monkey (241) showed this trypanosoma in its blood eight days after injection. At the time of writing this Report, the experiment was only just begun, but we are recording it because a hitherto undescribed filaria was discovered in this monkey's blood.* (See Plate 1.)

This was on November 12, 1903, on the day that the trypanosoma was also first seen in films. The filaria embryos had no sheath, a pointed tail, and were about 200 $\mu$ long (average of six measured, varying from 189 $\mu$ to 218 $\mu$). There was a clear space at the head, and the break in the continuity of the cells, which was well marked, was about 46 $\mu$ (limits, 43—50 $\mu$) from the head end.

*Guinea Pig.*—One guinea pig showed trypanosomata in its blood 29 days after injection. On November 17, 1903, when this Report was written, the animal was still alive and well, and showed parasites in its blood (nine weeks after injection).

*Rat* (Wild).—Parasites appeared in the blood five days after injection. Death occurred two days later.

*Goat* (Native).—Trypanosomata appeared in the blood 15 days after injection. During the disease the goat's temperature often rose to 107° F. The parasites were seen in the blood on November 11, 1903, and on November 17 (two months after injection) the animal was still in good condition.

*Sheep* (Native).—Trypanosomata seen in the blood 18 days after inoculation. In this animal the parasites were never very numerous, and the majority were small, vacuolated and deformed. The sheep's temperature was often 107° F. On November 17, 1903, trypanosomata were present in blood films, and the animal was still in good condition.

### (c) *A Trypanosoma occurring in a Dog sent to Entebbe from East Africa* (*Abyssinian Boundary Commission*) (T. V).

This variety of trypanosoma was obtained from a dog which had accompanied the Abyssinian Boundary Commission. It was stated that the Commission has lost on their journey, from a similar disease, the following animals:—Eleven Boran and Abyssinian ponies as well as several camels and five English dogs. None of their donkeys or mules, which were Abyssinian, suffered from the disease. The breed of this dog was half Airedale and half bull terrier. It was received on July 13, 1903, by Mr. R. J. Stordy, Principal Veterinary Officer to the Uganda and East Africa Protectorates, to whom we are indebted for the opportunity of studying the disease. On July 20, he noticed that the dog, who previously had been famed for his pluck, became very nervous. One month later, August 20, he found the trypanosoma in the dog's blood, having previously noticed that the corneæ were becoming opaque. On August 23, he despatched the dog to the Commission at Entebbe.

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* Since this Report was written originally, this experiment has been reported by Dr. Ross, see the 'Journal of Tropical Medicine,' January, 1904. At the time of discovery, Dr. Ross was assisting the Commission and had promised not to utilise any results obtained by us until published by the Royal Society.

A native (Abyssinian) dog, which was the companion of the English dog and had accompanied it throughout the expedition, remained quite healthy.

From the Boundary the route taken was from Lake Rudolph *viâ* Baringo to Nakuru. The journey from Baringo to Nakuru occupied four days. Two of the ponies above mentioned died of the disease at Nakuru, which indicates that probably the infection occurred at or near the Boundary, and certainly some distance north of Baringo.

### EXPERIMENT 160.—Dog (English).

This dog arrived in Entebbe on August 26, 1903, having been sent up from Naivasha, B.E.A., by Mr. Stordy. The animal is rather thin and has opacity of both corneæ, and is consequently blind.

August 27. Many trypanosomata found in the blood (T. V).

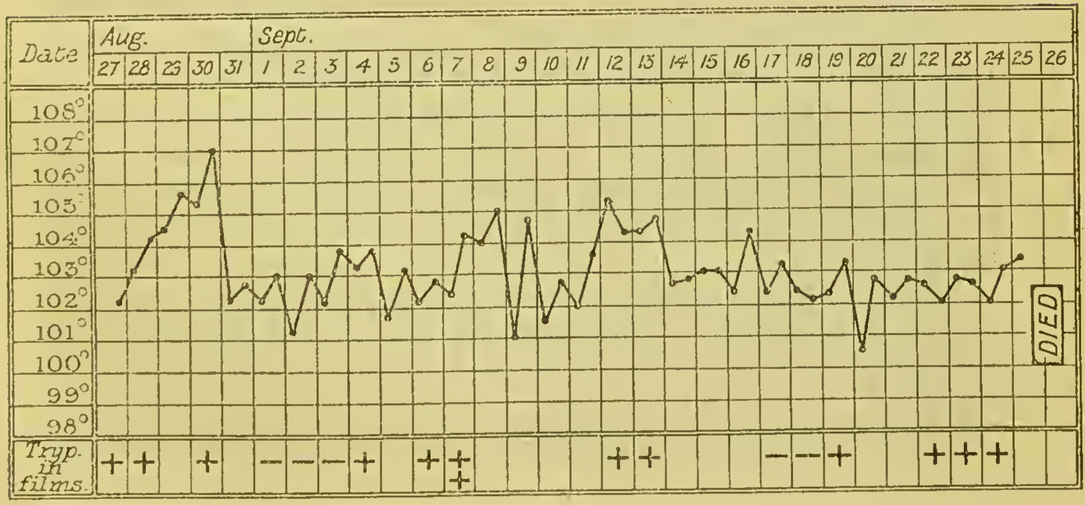
August 28. Many trypanosomata in the blood, which clotted readily. Three c.c. blood injected into Monkey 134.

September 3. During the past few days, since its temperature fell to 102°·4 on August 31, the animal lies about less.

September 7. Dog getting thinner. Blood swarming with trypanosomata.

September 12. Eyes are getting more prominent.

September 25. The dog is now in an emaciated condition, but no swelling of the legs or other part of the body has appeared. He is still able to stand and walk.



September 26. Dog died last evening.

The preceding chart (p. 23) shows the temperature curve and the presence or absence of trypanosomata in the blood from August 27, when the animal arrived in Entebbe.

September 26. *Post-mortem*, 12 hours after death.

Body much emaciated. Both corneæ opaque. No enlargement of superficial glands. No subcutaneous œdema anywhere. On opening the thorax no excess of pleural or pericardial fluid was present. Heart normal; no petechiæ or jelly-like material at the base. Both lungs had small embolic areas under the pleura, otherwise normal.

There was no increase of peritoneal fluid. The spleen was markedly enlarged, measuring 12 inches by 2½ inches broad. On section its substance was soft. The liver and kidneys were normal. There was some enlargement of the mesenteric and retroperitoneal glands.

### *The "East African" Trypanosoma* (T. V). *Its Morphology and Pathogenicity for Animals.*

In the blood of the naturally infected dog (Experiment 160) the parasites were long and slender, measuring in stained films from 20 to 30 μ in length, of which about 6 to 8 μ represented the flagellum. The posterior extremity was usually sharply pointed. On one occasion, when many parasites were present in the dog's blood, several paired forms were seen in which the posterior extremities were fused—the "conjugation" forms of Bradford and Plimmer (see Plate 2, *a*).

In a monkey (*Cercopithecus rufoviridis ?*) inoculated from this dog, the parasites were also long and slender, and on several occasions were found swarming in the blood.

Dogs, monkeys, guinea-pigs, rats, a baboon, bull, sheep, goat and donkey were injected with this trypanosoma. The results noted were as follows :—

*Dog.*—Trypanosomata appeared in the peripheral blood on the third and fifth day after injection. The former dog died 28 days after injection, the latter was alive 10 days after inoculation, when this Report was written. The dog (160), which had contracted the disease naturally, lived at least 72 days after the latest possible date of infection (July 13, when he was received by Mr. Stordy), though how long before this date infection actually occurred we had no means of ascertaining.

*Monkey.*—Three monkeys were inoculated. One (*Cercopithecus rufoviridis ?*) (No. 134) showed parasites in its blood five days after inoculation, and was still alive on November 17, 1903, 81 days after injection.

Another (Cercopithecus, black-faced) died of tuberculosis four days after inoculation without showing any trypanosomata in its blood, whilst the third monkey (also a black-faced Cercopithecus) showed the parasites in its blood on the third day.

*Guinea-Pig.*—Two guinea-pigs were inoculated. One died on the 10th day without having shown any parasites in its blood, the other was alive and well 43 days after injection, and showed no trypanosomata in its peripheral circulation.

*Rat (Wild).*—Two injected rats showed parasites on the third and seventh day, and died on the 10th and 14th day respectively after inoculation.

The *Baboon*, *Sheep*, *Goat*, *Bull* and *Donkey* were all alive and well 43 days after inoculation, and on no occasion could trypanosomata be discovered in films of their blood. We did not, however, inject their blood into rats to see whether it was infectious.

The chief symptom noted in the susceptible animals was irregular fever, the first rise of temperature often coinciding with the appearance of the trypanosomata in the peripheral blood. Sometimes, however, the first appearance of trypanosomata in the peripheral blood was unaccompanied by any rise of temperature. In Monkey 134 the temperature was sometimes as high as 107°·4 F., and was frequently at 106° F.

In some of the animals there was a rapid fall of hæmoglobin and wasting was marked. Œdema, enlargement of lymphatic glands and opacity of corneæ were absent.

*Post-mortem*, one dog had ecchymoses outside and in the interior of the heart and hæmorrhagic embolic areas in the lungs. Its spleen was enlarged and soft. There was no fluid in any of the serous eavities, nor were the lymphatic glands enlarged.

In two rats the only abnormal appearance *post-mortem* was *slight* enlargement of the spleen.

### (d) *A Trypanosoma Occurring in a Sick Mule in Entebbe* (T. VI).

#### Experiment 179.—Col. Sadler's Mule.

This animal had been in Africa about five years. At first it was in the "fly-belt" of East Africa. It had been in Uganda about 18 months and remained well until it had fever and swelling of the glands in the groin. It was first noticed to be sick on July 3, 1903.

September 9, 1903. Animal brought to the laboratory. Had fever and slight swelling of glands in groin, none elsewhere. Ate well. Was getting thin.

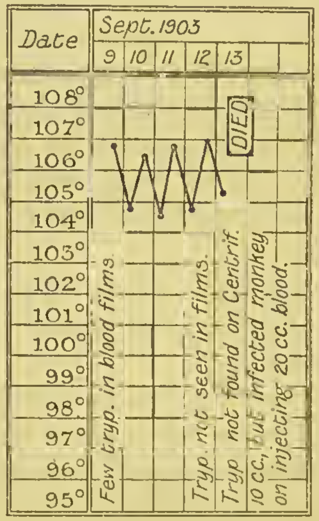
A film of blood was examined and showed a few trypanosomata, very vacuolated in appearance. Eighteen c.c. of blood were injected into Dog 167.

September 12. Blood very pale and watery. *No tryponosomata seen in films.*

September 13. The animal was brought to the laboratory this morning and, on its way, fell down and was unable to rise again. Its breathing was very hurried.

A blood film was examined but *no trypanosoma found.* It died at 11 A.M. and a *post-mortem* was made half an hour after. Blood was obtained from the jugular vein and inoculated into two monkeys (180 and 181) and a guinea-pig. Both monkeys developed trypanosomata in their blood. Ten c.c. of the blood was centrifuged about one and a half hours after death, but no trypanosomata could be found.

The accompanying chart shows the temperature curve and the presence or absence of trypanosoma in the blood:—



September 13. *Post-mortem* half an hour after death.

External appearance—nothing abnormal. No enlarged glands could be felt. No fluid in pericardial or pleural cavities. A few small petechiæ were present on the external surface of the heart and one small one in the interior of the right ventricle. The blood was very watery and the heart muscle pale. No jelly-like material round base of heart. Lungs normal. Spleen enlarged and weighed 4½ lbs. (normal said to be 2 lbs.); substance firm. Liver somewhat enlarged, weighing 14 lbs., substance congested. Kidneys normal. There were no enlarged glands in mesentery or retroperitoneum.

*Remarks.*—The interesting feature of this experiment is the rapidity with which the trypanosomata disappeared from the animal's peripheral blood during the last few days of its life. On September 9 several trypanosomata were seen in a blood film. Three days afterwards they could not be found in blood films, and on the following day they were so scarce that they could not be detected in 10 c.c. of centrifuged blood taken shortly after death. That they were present, however, is proved by the fact that two of the three animals injected with the blood, obtained *post-mortem*, developed trypanosomiasis.

### *The "Entebbe Mule" Trypanosoma* (T. VI).—*Its Morphology and Pathogenicity for Animals.*

When the mule's blood was first examined, four days before death, only a few trypanosomata were found. These were rather short and very vacuolated. On injection into a dog (167) the parasites which appeared in its blood were long and thin. In another dog (197) which was injected with blood from Monkey 180, the average length of the trypanosoma was about 27 μ, the flagellum measuring 7—10 μ. The edge of the undulating membrane was very distinct and the posterior extremity squarely blunt, resembling a truncated cone. One sexual (?) form seen in a film was 17 μ long (flagellum 6 μ) and in its widest part measured nearly 5 μ across (see Plate 2, *b*). In another dog (226) which was injected from the same monkey (180), but at a later date, the parasites were shorter and broader, sometimes vacuolated and with a short flagellum. In inoculated monkeys similar variations in size and form were observed. In Monkey 180 on several occasions the trypanosomata were normal in appearance, but on other occasions they were crescentic in shape with a broad thick body and very short flagellum. The protoplasm was very vacuolated and in many respects the parasites resembled those seen originally in the blood of the mule. It was noticed that when the parasites showed this peculiar appearance, trypanosomata were, as a rule, scanty in the animal's blood. Blood stained two or three days later contained normal parasites (see Plate 2).

In Monkey 181, the trypanosomata were normal in appearance, thin, about 18 μ long and with a short flagellum. Two days later larger and longer forms were seen (27 μ long) with vacuolation of the protoplasm. As in Dog 197, the posterior end was squarely blunt with the centrosome a long distance from the tip (3—4 μ). Six days later, the trypanosomata were very scanty in the blood and presented the same vacuolation and deformity of shape as Monkey 180. Two days later, many normal parasites were again seen, others still showing vacuolation. On the whole, the parasites seen in the blood of the monkeys were smaller than those in the dog, the flagellum especially being shorter. Dogs, monkeys, rats, a guinea-pig, sheep, goat, ox, donkey, baboon and jackal were inoculated with this trypanosoma, and with the following results:—

*Dog.*—Trypanosomata appeared in the peripheral blood 4, 5, 8 (twice) and 9 days after injection and the dogs died on the 10th, 14th, 26th, 15th and 10th day respectively. It should be added that the last animal had many ankylostomata in its intestine, which were the immediate cause of death.

*Monkey.*—Four monkeys were inoculated, two showing parasites on the fourth day and two on the seventh day. Death occurred in 8, 12, 21 and 40 days respectively, but in every case death was due to some other cause than the trypanosoma infection.

*Rat (Wild).*—Trypanosomata appeared in the blood on the third and fifth day. The former animal died in 22 days, the latter was still alive 7½ weeks after the inoculation, when this Report was written. This trypanosoma appears to be less virulent for rats than either the "Jinja" or the "East African" parasite.

*Jackal.*—The infected jackal was an interesting experiment. On July 24, 1903, it was injected with blood containing human trypanosomata from Karala Barigi. The parasite appeared in its blood on August 4 and continued present, with intervals of absence, until September 22, after which date human trypanosomata were no longer seen in films. On the night of November 1, the jackal killed and devoured almost entirely a monkey which had many "mule" trypanosomata in its blood. On November 3, about 36 *hours* after its feed, the blood was found to contain very many trypanosomata. On November 5 the jackal looked seedy and lay about a great deal. There was a semi-purulent discharge from the eyes. Trypanosomata continued present in the blood and on November 17 were swarming. The jackal died on that date, about 15 days after it ate the monkey.

Three interesting points are brought out in this experiment. First it shows that the jackal resembles the dog in its behaviour to the human trypanosoma, inasmuch as the parasite multiplies to a certain extent in the blood but eventually disappears or becomes so scanty as not to be detectable in blood films. Secondly, that the animal was susceptible to infection with another trypanosoma which proved fatal, showing that the human and mule trypanosomata are different. Thirdly it shows that infection can be conveyed *viâ* the alimentary tract, though possibly not through the stomach but through abrasions of the mouth or other soft parts, produced, in this case, by the bones of the monkey whilst being eaten or swallowed.

The inoculated guinea-pig, sheep, goat, ox, donkey and baboon were all alive and well on November 20, 1903, 69, 53, 53, 53, 38 and 38 days respectively after inoculation. The animals were all reinjected with blood from the jackal, containing many trypanosomata, on November 8 and 9, but on no occasion could the parasite be found in blood films. Nevertheless they all suffered from occasional rises of temperature, sometimes as much as 105° or 106° being recorded, and the ox got distinctly thinner. We injected some blood from this ox (202) into a dog (239) and although the ox never showed trypanosomata in blood films, its blood was infectious, for the dog had parasites in *its* blood eight days after the injection and died a week later. We did not test the blood of the other animals, but had we done so, in all probability we should have found that it was infectious, seeing that all the animals had irregular rises of temperature.

Of the susceptible animals, the dogs and monkeys had fever and the latter, in addition, showed diminution of hæmoglobin and wasting. Œdema, enlargement of superficial glands and opacity of corneæ were absent. In the dogs, the disease was rather acute and none of them showed much wasting.

*Post-mortem.*—The spleen was enlarged in all the animals, especially in the dogs. The liver and kidneys were often pale and fatty-looking. The heart rarely showed petechiæ and only occasionally was there any free fluid in the serous cavities. Two of the dogs had embolic areas in the lungs. In the rat, the abdominal and retroperitoneal glands were considerably enlarged; none of the other animals had any notable enlargement of the lymphatie glands. The rat's spleen was enlarged, and smears showed, in addition to many normal trypanosomata, vacuolated and "amœboid" forms.

## Section II.—CONTINUATION OF THE OBSERVATIONS ON FIVE CASES OF TRYPANOSOMIASIS IN MAN, AND OF THE EARLIER EXPERIMENTS ON MONKEYS AND OTHER ANIMALS, INCLUDING SOME OBSERVATIONS ON PIROPLASMOSIS IN DOGS.

Of the five cases of Trypanosomiasis originally under the observation of the Commission, four were apparently in good health at the time of writing this Report, the fifth (Bara Risgallah) on one occasion was found asleep when at duty. We failed to find trypanosomata in the cerebro-spinal fluid of any of the patients, even in the case of J. Murjan and Karala Barigi, both of whom had shown them on two occasions previously. On centrifuging 10 c.c. of blood from these five patients, only two of them (Murjan and Barigi) showed trypanosomata in November, 1903.

Of the earlier experiments, we are furnishing records only of those which are completed or which have a special interest. The details of these experiments prior to August 27 will be found in Nos. I and IV of the Reports of the Sleeping Sickness Commission already published.

Monkeys 34 and 95, which have been injected with cerebro-spinal fluid containing trypanosomata, have died. The details are as follows:—

### EXPERIMENT 34.—Monkey (*Macacus rhesus*).

Injected with C. sp. fluid (containing T. I) in vertebral canal on April 8, 1903.

Last note by Colonel Bruce was on August 25.

September 7. During the past 10 days, this monkey has been lying about much and often sitting with his head bent down on his chest. Yesterday he was much as usual, but got rapidly worse, and was obviously dying this morning. His temperature was 94°·4.

*Post-mortem* immediately after death.

Body thin. Coat very rough and staring. Heart normal. In the lungs there were a few pigmented areas looking like old tubercles. The liver was fatty. Spleen normal. Kidneys pale. The inguinal, mesenteric and retro-peritoneal glands were enlarged, also those in the left axilla.

*Brain.*—No very marked signs of disease. Not much flattening of the convolutions. Slight adhesions at the base of the brain. There was a little opacity of the sulci.

Some fluid from the third ventricle was examined, and a few living and active trypanosomata were seen. The film contained much blood, and the trypanosomata may have come from the blood.

Films of heart blood stained, showed very few trypanosomata, but no "involution" forms.

The brain was fixed and hardened and sent home to Dr. Mott. We have since heard that no minute pathological changes were present in the brain of this monkey.

### EXPERIMENT 95.—Monkey (*Cercopithecus* sp.).

Injected intraspinally with C. sp. fluid (containing T. I) on May 14, 1903. Tryp. in blood films on June 4.

August 26. About 8.30 A.M. monkey had a convulsive seizure affecting the left side of the body. It lay on the ground for about an hour suffering from paresis. This gradually passed off. Monkey appears rather bent up.

August 27. Evening temperature, 101°·6.

August 28. Monkey getting gradually weaker. No definite symptoms of Sleeping Sickness.

*Blood examined.* Animal very anæmic; blood obtained with great difficulty, and is extremely pale. *Tryp. abs.* Mal. absent. Many nucleated red corps. Temperature: morning, 100°; evening, 100°·8.

August 29. Animal was found dead this norning.

*Post-mortem, Brain.*—Convolutions a little flattened; not congested, and no ground-glass appearance as in Sleeping Sickness.

*Heart.*—No jelly-like material externally. No petechiæ.

*Lungs.*—Both showed embolic areas.

*Spleen.*—Congested and slightly enlarged.

*Kidneys and Liver* pale.

A drop of C. sp. fluid from lateral ventricle examined, but no living trypanosomata seen. Possibly the animal had been dead too long.

Heart blood film:—No tryp. seen in stained preparation.

### "FRESH-FLY" FEEDING EXPERIMENTS.

The two "fresh-fly" feeding experiments next recorded are of considerable interest.

### EXPERIMENT 136.—Monkey (*Cercopithecus* sp.). Small.

This animal was kept as a "control" from July 23 until August 28, 1903. It was then used to feed fresh tsetse flies upon. Between August 29 and October 4, 723 flies fed on this monkey.

October 6. During the past few days, this monkey has been looking seedy, sitting crouched up with its back bent and head thrown forward on its chest, much like the attitude adopted by the sick monkeys, 1, 60 and 34 before they died. He has been getting very thin, evidently the drain of blood, by so many flies feeding on it, has been too great. Died at 3 P.M. to-day.

*Post-mortem.*—All organs were very pale and bloodless. Film of heart blood was typical of profound anæmia—red corpuscles of all sizes, many nucleated and showing polychromatophilia.

*No trypanosomata* seen.

*Remarks.*—Although 723 "fresh" flies fed on this monkey, trypanosomata were never found in its blood. The original experiment (No. 94, in earlier Reports) had only 225 flies fed on it, when the tryp. appeared in its blood. The flies were brought sometimes from the Botanical Gardens, and at other times from the shore of the lake near the hut-tax labourers' camps, as they had always been. It is significant that over 700 flies failed to convey the tryp. to this monkey—although it was small, about the same size as Monkey 94. The only obvious difference between this experiment and Experiment 94 (as recorded in No. I of the Sleeping Sickness Commission Reports) is that during the progress of this experiment, practically no hut-tax labourers came into Entebbe, and those that did come were housed away from the fly-area.

### EXPERIMENT 228.—Monkey (*Cercopithecus* sp.).

Between October 12 and November 12, 1903, no less than 757 freshly caught tsetse flies were fed on this monkey without causing the appearance of trypanosomata in its blood. The same remarks as to hut-tax labourers apply to this experiment as to Experiment 136.

### (1) *Re-Injection Experiments.*

In order to ascertain whether "refractory" animals could be made more susceptible and show the trypanosoma in the peripheral blood, we re-injected them with trypanosoma-containing blood or cerebro-spinal fluid, sometimes on as many as three occasions at intervals of a week or a fortnight, and in every case with a negative result.

The details, in tabular form, are as follows :—

Blood Trypanosoma.

| | |
|---|---|
| Guinea-pig 81 ............ | Re-injected once. |
| Goat 90 .................. | ,, once. |
| Donkey 101 ............ | ,, twice. |
| Ox 148 .................. | ,, twice. |
| Sheep 149 ............... | ,, once. |
| Pup 146 .................. | ,, once. |

Cerebro-Spinal Fluid Trypanosoma.

| | |
|---|---|
| Guinea-pig 82 ......... | Re-injected once. |
| Sheep 89 ............... | ,, four times. |
| Donkey 100 ......... | ,, three times. |
| Ox 132 ............... | ,, three times. |
| Pup 144 ............... | ,, three times. |

Guinea-pig 82 became pregnant after the second injection, and bore a healthy young one which thrived.

The pups 144 and 146 both developed the *Piroplasma Canis* in their blood, to which infection they succumbed. These experiments are given more in detail later. An interesting fact is that the blood of Pup 144, although it never showed the trypanosoma in films, was nevertheless infectious, because a monkey inoculated with it developed trypanosomata as well as piroplasmata in its blood.

We also inoculated a cat with 10 c.c. blood containing the human trypanosoma, but eight weeks after the inoculation the cat's blood did not show any parasites in films.

### (2) *Re-inoculation and Cross-inoculation Experiments with the Human Blood and Cerebro-spinal Trypanosomata.*

Several of the earlier experimental monkeys having failed to show trypanosomata in blood films at many of the weekly routine examinations of their blood (the parasites having previously been easily found in them), it occurred to us that possibly a condition of immunity had been established. We therefore re-injected five of these monkeys, Nos. 32, 61 and 123 with blood, and Nos. 54 and 96 with cerebro-spinal fluid containing trypanosomata. In one case the parasite did not reappear in the peripheral circulation as long as six weeks after this re-injection. In the other cases, sufficient time had not elapsed when this Report was written, to be certain that the trypanosomata would not reappear, though in none of the cases had they done so. These observations seem to indicate that the monkeys in question had become immunised against the trypanosoma injected. The next step, should the first trypanosoma have failed to reappear, would be to inoculate Monkeys 54 and 96 with the blood trypanosoma, and Monkeys 32, 61 and 123 with the cerebro-spinal trypanosoma. It was arranged that one of us (E. D. W. G.) should continue these experiments on those lines, because if the immunised monkeys failed to respond to injections of the other trypanosoma, this would be in favour of the identity of the *Trypanosoma Gambiense* and the *Trypanosoma Ugandense.*

The two cases next reported are Sleeping Sickness in a Persian, this being the first instance of the disease in an Asiatic; a case of "recurrent fever," associated with the presence of spirochætes in the blood, sent up as a case of Sleeping Sickness.

(3) *Case* 248. *Sleeping Sickness in a Persian. Name, Suleman Bin Mohamed. Age about* 45 *years. Came originally from Bushire, on Persian Gulf.*

October 28.—*History.* Has been in Africa 20 years, of which he spent the first two years in East Africa. Has been in Uganda on and off for 18 years, and has been down to the Coast twice in that time. Latterly he has been a "headman" in the Government Transport Department, and when in Entebbe has lived near the lake. In fact, for some time he has lived almost like the natives and with them. He became ill two months ago, and was then seen by Dr. Hodges, whose report is as follows:—

September 2. There are general tremors, anæmia, slight pyrexia. Temperature, 100° F. Weak, irregular, rapid pulse. Spleen slightly enlarged. Left otitis media. Blood films examined. Malaria found.

October 28. *Lumbar puncture performed.* Tryp. numerous; one seen in first field. Many lymphocytes. No R.B.C.

October 31.—*Present state.* Patient is a very tall man, obviously wasted and ill. There is a heavy, sad look about the face and eyes. General condition very weak and feeble. Gait weak and staggering. Voice weak. Marked tremor of tongue and lips. Slight tremors of fingers and hands. There is a fairly marked general tremor of the body and head. No enlarged glands about neck, slight in groin (due to sore feet). Has pain in left ear, due to otitis media. Pulse, fair tension, 68 per minute. K.J. sluggish. Heart sounds feeble; no bruit.

November 6. Photograph taken by Mr. R. J. Stordy.

November 12. Patient has improved to a certain extent since he has been in hospital.

(4) *Bafirawala. Æt.* 12 *years. Recurrent (Spirochæte) Fever.*

This patient was admitted to hospital on August 12, 1903, as a case of Sleeping Sickness. He never showed any definite signs of

Sleeping Sickness, but was suffering from "Spirochæte"—(? Relapsing)—Fever; and as this appears to be the first case occurring in Uganda which has been studied, we thought it advisable to put it on record.

August 18. During the past four days the temperature has risen gradually until it reached a maximum of 104°·8 last evening. This morning it fell to 103°·4. Patient complains of pain in both knee joints, but has no pain elsewhere. Pulse is rapid, but regular in force and rhythm. There is a slight systolic apical murmur. The spleen is considerably enlarged, and can be felt two fingers' breadth below the costal margin.

Blood films were examined, and some spirochætes were seen; also filariæ. No trypanosoma or malaria. A fresh drop of blood was examined later, but no motile parasites were seen.

At 6 P.M. the temperature rose to 106°·2 F.

August 19. This morning the temperature has fallen to 99°·4, and patient is more comfortable. The spleen is smaller than it was yesterday. Tr. opii is being applied to the swollen, painful knee joints.

August 20. Patient's general condition is better than yesterday, but the pain in the knees continues.

August 23. Temperature rose again yesterday to 102°·4, and this morning is 103°·8. The spleen is easily palpable, but no larger than yesterday. Blood examined; no spirochætes, but one doubtful malarial parasite seen. Temperature at 3 P.M. was 99°. Blood again examined; no spirochætes or malaria seen.

August 26. Temperature rose again last night to 104°·4, and this morning has fallen to 101°·2. Blood examined: few spirochætes seen.

September 14. Since last note patient has had slight fever, with occasional rises to 101°.

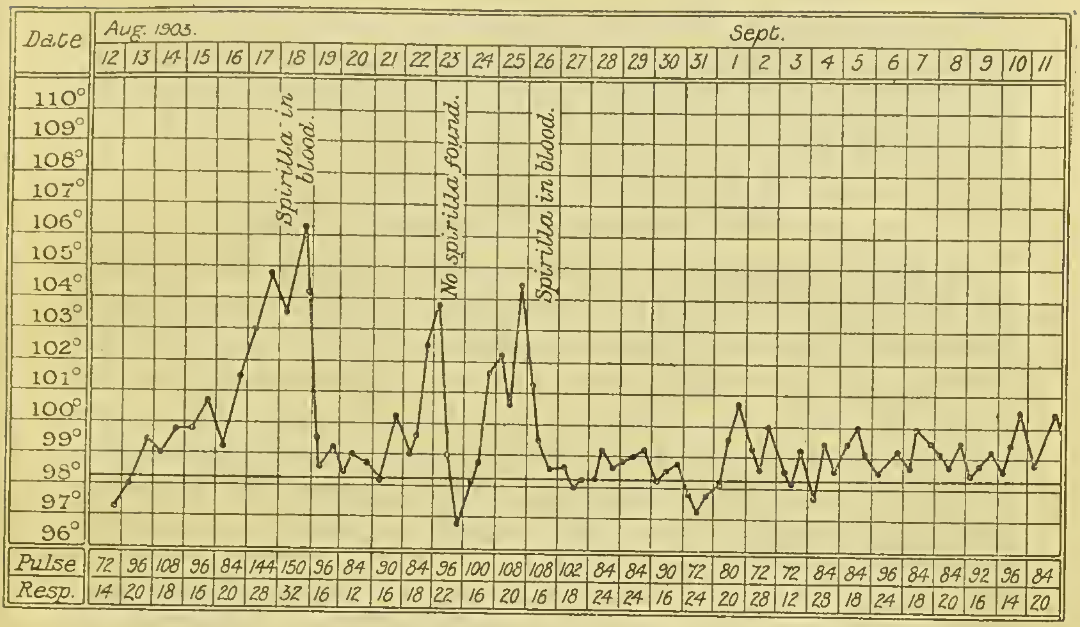
To-day *lumbar puncture* was performed, but no fluid could be obtained, indicating that there was probably no increased pressure in the spinal canal.

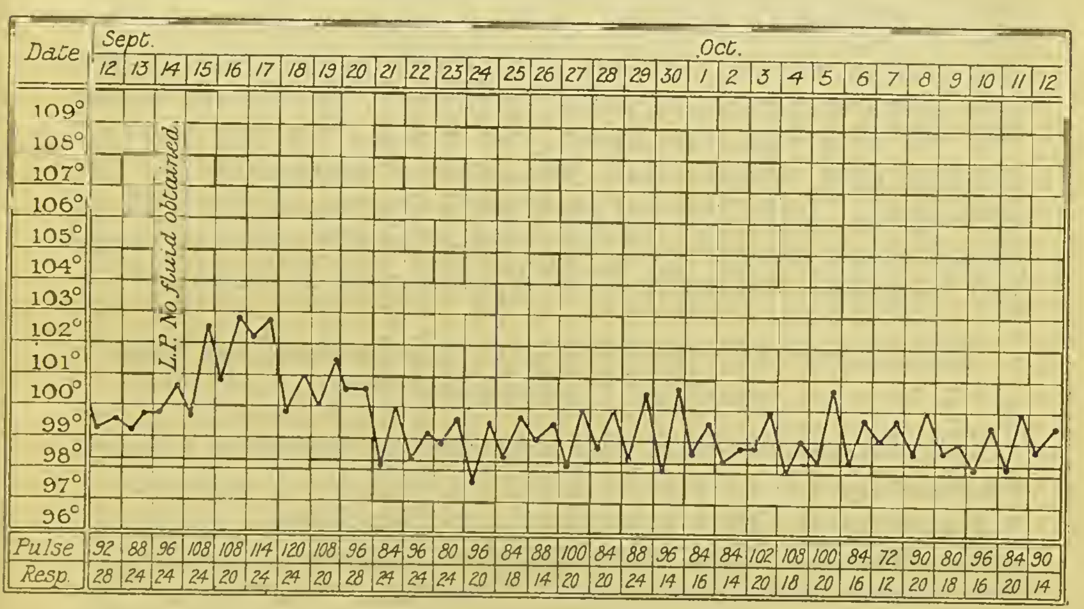
Patient has headache, and his speech is weak and indistinct. No tremor of the tongue or fingers. The gait is rather weak, but probably this is on account of the swelling and pain in the knees.

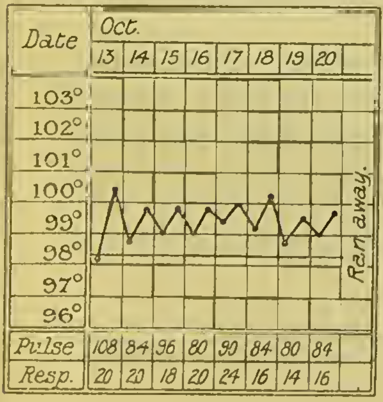
October 20. The temperature was raised for a few days after the lumbar puncture, and there has been slight pyrexia since last note.

Patient's general condition is improved. He ran away from hospital to-day.

The following chart shows the temperature curve, the pulse and respiration rate, and the presence or absence of spirochætes in the blood:—


Date
Aug. 1903.
Sept.
12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11
110° 109° 108° 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
Spirilla in blood.
No spirilla found.
Spirilla in blood.
Pulse 72 96 108 96 84 144 150 96 84 90 84 96 100 108 108 102 84 84 90 72 80 72 72 84 84 96 84 84 92 96 84
Resp. 14 20 18 16 20 28 32 16 12 16 18 22 16 20 16 18 24 24 16 24 20 28 12 28 18 24 18 20 16 14 20


Date
Sept.
Oct.
12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12
109° 108° 107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
L.P. No fluid obtained.
Pulse 92 88 96 108 108 114 120 108 96 84 96 80 96 84 88 100 84 88 96 84 84 102 108 100 84 72 90 80 96 84 90
Resp. 28 24 24 24 20 24 24 20 28 24 24 24 20 18 14 20 20 24 14 16 14 20 18 20 16 12 20 18 16 20 14


Date
Oct.
13 14 15 16 17 18 19 20
103° 102° 101° 100° 99° 98° 97° 96°
Ran away.
Pulse 108 84 96 80 90 84 80 84
Resp. 20 20 18 20 24 16 14 16

Since writing the above, Dr. Hodges has had several cases of a similar kind in Entebbe, occurring in an Indian and in native Waganda. In all his cases the spirochætes were very scanty in the blood and were seen only in stained films, not in a fresh specimen.

### (5) *Piroplasmosis in Two Experimental Dogs.*

As mentioned previously in this section, two pups (144 and 146) received respectively four and two injections of human trypanosomata without showing the parasites in blood films. A week or two after the last injection both animals were seedy, and on examining their blood an infection with the *Piroplasma canis* was discovered. The puppies had the run of the laboratory compound, and occasionally strayed beyond its precincts, so that they may have become infected outside, especially as two other fatal cases occurring in Entebbe were brought to our notice about the same time.

A monkey (213) and a dog (214) were injected with blood from Pup 144. Both monkey and dog showed piroplasmata in blood films on the 11th day, the monkey also showing trypanosomata on the 15th day, although the pup itself had never shown trypanosomata in films, even after four injections of cerebro-spinal fluid. The injected monkey was still alive when this Report was written; the dog developed irregular fever, suffered rapid diminution of hæmoglobin, and had albuminuria. It died five and a-half weeks after inoculation with the piroplasma, and *post-mortem* the liver, spleen and kidneys were enlarged and congested. The details of the experiments are as follows :—

### EXPERIMENT 144.—Pup (Brindled). Injected with C.-sp. Fluid containing Trypanosomata (T. I).

This dog was reinjected with C.-sp. fluid on September 1 (4 c.c.), on September 7 (4 c.c.), and on September 14 (8 c.c.).

September 15, 1903. Tryp. absent from blood films.

September 22. Tryp. absent from blood films.

September 29. Tryp. absent. *Piroplasma canis* present in large numbers. No symptoms except rise of temperature (105°·8). Urine normal in appearance.

October 1. Blood very pale to-day. Animal worse. Piroplasmata numerous. At 4 P.M. she passed a little urine, markedly coloured by hæmoglobin. Much albumen present. Some blood was taken and injected into Monkey 213 and Dog 214. The monkey developed the *piroplasma in its blood on the* 11*th day* and *trypanosoma* 15 *days after injection.* The dog (214) became infected with the piroplasma and died about five weeks later.

October 2. Animal died during the night.

*Post-mortem.*—Body fairly well nourished. No enlarged superficial glands. No increase of pericardial or pleural fluid. Heart normal. Lungs showed minute embolic areas. Liver somewhat enlarged and rather fatty. Spleen enlarged, measuring 7 inches by 2 inches. Kidneys were markedly congested, especially the cortex and the surface. The hilum was red in colour as though stained with hæmoglobin. Bladder contained reddish brown (dark sherry-coloured) urine. No increase of peritoneal fluid.

*Remarks.*—This puppy harboured the trypanosoma, although it failed to show them in blood films. On injecting the puppy's blood into a monkey, the trypanosoma appeared in 15 days in the peripheral blood.

### EXPERIMENT 146.—Puppy. Injected with Human Native Blood, containing Trypanosomata (T. II).

September 21, 1903. Reinjected 5 c.c. blood from J. Murjan containing trypanosomata.

September 22. Tryp. absent.

September 29. Tryp. absent. *Piroplasma canis* present (fewer than in Dog 144). Blood pale and watery, but dog seems in good condition. Some fever. Urine normal.

October 13. The dog is somewhat out of condition, but no dark urine has been observed, and he takes his food fairly well.

November 3. The temperature rose on October 27 to 106°, and since then has been fluctuating between 103° and 104°·6. The animal has been lying about again for the last few days. During the night he passed some "smoky" urine, containing albumen. Dog looks very thin and seedy to-day. Piroplasma present in the blood.

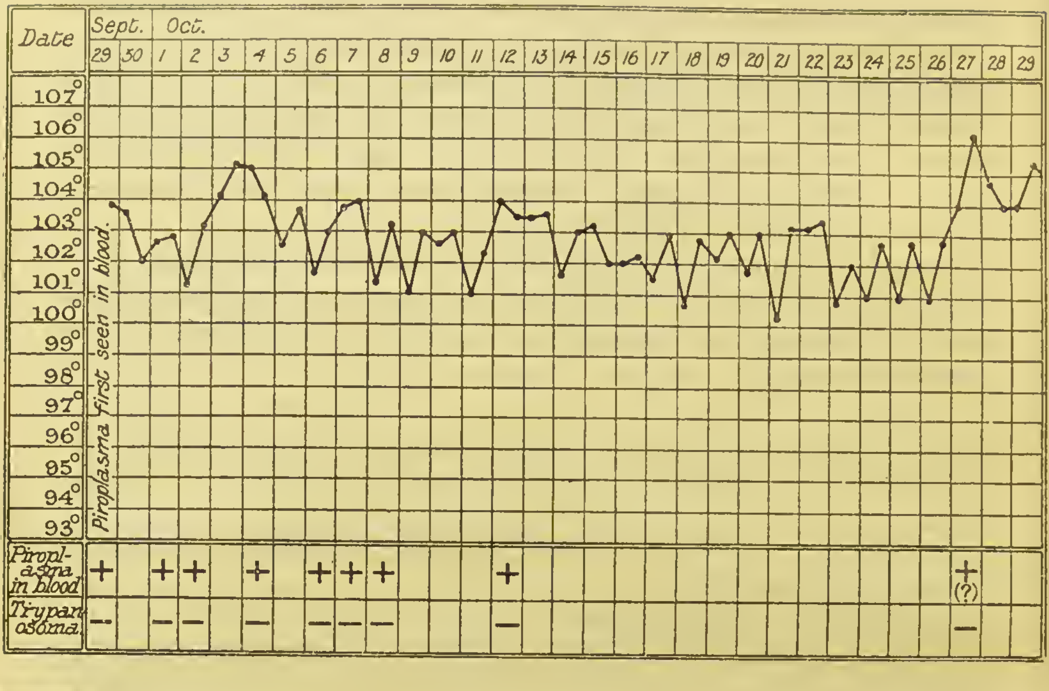
November 5. Animal died to-day at 2 P.M.

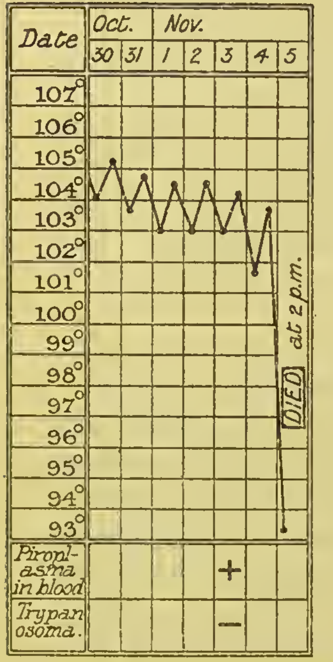
The following chart (p. 38) shows the temperature curve and the presence or absence of trypanosoma and piroplasma in the blood.

November 5. *Post-mortem.*—Soon after death. Body fairly well nourished. No enlarged glands. Heart normal. Both lungs show well-marked embolic areas. Spleen enlarged, measuring 10 inches by 2½ inches. Liver congested. Kidneys look normal—capsule strips readily. The bladder contains some urine containing blood and albumen.

*Smear of Lung Emboli.*—Many red corpuscles infected with piroplasma, many more than were found in the peripheral circulation.

*Smear of Spleen.*—Some infected corpuscles present.





## Section III.—Is there a Specific Carrier for Each Variety of Trypanosoma?

In the "Appendix to the Further Report on the Tsetse Fly Disease in Zululand," Colonel Bruce states that he examined the proboscis and stomach contents of the tsetse flies at varying intervals after feeding on an infected animal. The proboscis showed one or two active trypanosomata up to 46 hours after feeding. The stomach, on the other hand, in some cases contained blood up to 118 hours after feeding, and in rare instances numerous and vigorously active trypanosomata were seen amongst the unchanged red blood corpuscles.

In our earlier experiments with the East African trypanosoma (T. V) in *Glossina palpalis*, we found that the trypanosomata very rapidly lost their motility in the flies' stomachs—sometimes in as short a time as three hours.

The "Jinja" trypanosoma (T. IV), on the other hand, was found actively motile in the stomach contents of *Glossina palpalis* after a considerably longer interval, up to 100 hours.

These observations naturally suggested the question "Is there a specific carrier for each variety of trypanosoma?"

In attempting to answer this question the following experiments were carried out:—

(1) *Glossina palpalis* was fed on animals infected with trypanosomata (T. IV, V and VI) and, after intervals of 6 and 24 hours, on healthy animals.

(2) Stomoxys were similarly fed on infected and afterwards on healthy animals. Tabanidæ were also tried, but it was found that they would not feed on dogs, monkeys or oxen when in captivity in feeding-boxes.

(3) *Glossina palpalis* and Stomoxys were fed on animals infected with trypanosomata (T. I, IV, V and VI) and dissected after varying intervals, in order to ascertain whether there was any striking difference in the length of time that the trypanosomata remained alive in the stomach contents.

The dissection experiments with the human trypanosoma are difficult, because one rarely finds many trypanosomata in an infected monkey, and consequently the stomach contents of flies contain comparatively few parasites.

The experiments conducted in East Africa (*vide* Section IV) show that one or more of the varieties of Glossinæ found there are capable of conveying the trypanosoma of Sleeping Sickness.

### (1) *Can the* Glossina Palpalis *Carry other Trypanosomata than the Human?*

The *Glossina palpalis* was fed on animals infected with the Jinga (T. IV), East African (T. V), and Entebbe mule (T. VI) trypanosomata, and, after intervals of 6 and 24 hours, on healthy monkeys. *Two of these experiments were positive.* The *Glossina palpalis* conveyed the "Jinja" trypanosoma six hours, and the "mule" trypanosoma 24 hours, after feeding on an infected animal. Negative results have so far been obtained in the case of T. V, the "East African" trypanosoma.

We would add that in order to eliminate as far as possible the chances of the "fresh" flies harbouring trypanosomata, the freshly caught flies were kept for 18 to 24 hours, then fed on a "clean" monkey, and then kept for another 24 hours before being used for the feeding experiments.

It was noticed in these feeding experiments (both with human and animal trypanosomata) that when monkeys became infected in this way, the parasites were never plentiful in the peripheral blood, even many weeks after infection occurred. The temperature, also, of monkeys infected by feeding did not show the same elevations as in the case of monkeys which developed trypanosomiasis after injection.

### (2) *Can other Biting Flies Convey Trypanosomata?*

Stomoxys and Tabanidæ were used in the attempts to answer this question. Stomoxys were easily procurable in Entebbe, especially where many dogs and cattle were herded. Mr. E. E. Austen informs us that there were at least two species of Stomoxys amongst those used in our experiments. Feeding experiments were carried out with all three animal trypanosomata (T. IV, V and VI), and *in no case*, so far, have we succeeded in conveying the parasites from infected to healthy animals by feeding Stomoxys on them after intervals of 6 or 24 hours.

Tabanidæ were less easy to obtain, and, moreover, were found useless for these feeding experiments, as they refused to bite dogs, monkeys or cattle when in captivity in the feeding-boxes.

### (3) *Dissections of Flies* (Glossina *and* Stomoxys) *after Feeding on Animals Infected with Trypanosomata.*

The results of these experiments are best seen in tabulated form as follows:—

Table II.—*Glossina palpalis.*

| Species of trypanosoma. | Longest time since feeding that active tryp. were seen in stomach contents. | Remarks. |
|---|---|---|
| | hours. | |
| Sleeping Sickness (T. I) … | 71 | Fairly actively motile |
| Jinja disease (T. IV) …… | 100 | Actively motile |
| East African (T. V) …… | 5½ | Movements very sluggish 3½ hrs. after feeding. Ceased after 5½ hrs. |
| Mule (T. VI) …………… | 20 | Only sluggishly motile at this time. Many inactive ones seen 21 hrs. after feeding |

Table III.—Stomoxys.

| Species of trypanosoma. | Longest time since feeding that active tryp. were seen in stomach contents. | Remarks. |
|---|---|---|
| | hours. | |
| Jinja disease (T. IV) ...... | 24 | Tryp. sluggishly active. Many active filaria embryoes seen* |
| East African (T. V) ...... | 12 | Some fairly active. Many inactive seen 21 and 27 hrs. after feeding |
| Mule (T. VI) ............... | 30 | Some inactive tryp. seen 43 hrs. after feeding |

* The monkey (241) on which these flies were fed had many embryo filariæ in its blood (see p. 22).

Several interesting points are brought out in these dissection experiments. First, the great variation in the times after feeding that active trypanosomata were seen in the stomach contents of *Glossina palpalis*, namely, 5½, 20, 71 and 100 hours. It explains the failure to convey the "East African" trypanosoma by feeding flies on infected animals, and 6 or 24 hours later on healthy animals as recorded on p. 39. It is curious that in the case of the "mule" trypanosoma, the fly-feeding experiment was positive after a 24-hour interval, although on dissection we did not find active trypanosomata in the flies' stomachs longer than 20 hours after a feed.

Secondly, in Stomoxys, the intervals after feeding, during which active trypanosomata were present in the stomach were more nearly the same in the case of the three animal trypanosomata (T. IV, V and VI), but it is noteworthy that the East African and mule parasites were found active for a longer period in Stomoxys than in *Glossina palpalis.*

Thirdly, in the earlier dissections *cold* physiological saline solution was added to the (often very viscid) stomach contents in order to get satisfactory fresh films. Later, we always diluted the stomach contents with *warm* saline, as it was found that by so doing the trypanosomata were active for longer periods after feeding; and all the times recorded in the above tables were got by that method.

The proboscis of the flies was also examined in many cases, but active trypanosomata were only occasionally found in Glossinæ, namely, 3¾ hours in the case of T. IV (Jinja), and 2½ hours in the case of T. V and T. VI (East African and mule). Although the proboscis was frequently examined at later periods, trypanosomata were never seen.

In the Stomoxys, the proboscis is darker and more opaque than in the Glossinæ, so that it is very difficult to see red blood corpuscles or trypanosomata in it. We therefore soon gave up looking for trypanosomata in the proboscis of Stomoxys.

These experiments were not quite completed when this Report was written, so that the times recorded above may have to be slightly altered as the result of further dissections.

In order to ascertain whether any developmental changes occur in the flies' stomachs analogous to those which are found in the case of the malarial parasite in the stomach of Anopheles, specimens of the stomach contents were stained (by Leishman's method) at varying periods after a feed. We may state at the outset that hitherto we have obtained no undoubted evidence of any such change.

In the case of the "Jinja" trypanosoma, Glossinæ were dissected and the stomach blood stained 42, 71 and 100 hours after a feed. In the first film (42 hours) the trypanosomata were normal in form and size, the protoplasm of the body vacuolated and staining fairly well, whereas the undulating membrane and flagellum were unstained. The nucleus and centrosome were stained red, and the latter was usually further from the posterior extremity than is normally the case.

In the film stained 71 hours after a feed, the trypanosomata were very numerous. This was noticed also in the fresh film which was examined at the same time. The parasites were all very long, and all stained badly. The protoplasm of the body was pink instead of blue; the undulating membrane and flagellum were unstained, as was also the nucleus in most cases. The centrosome, on the other hand, was deeply stained.

No trypanosomata could be seen in the stained specimen made 100 hours after a feed, though in the fresh film active trypanosomata were present.

In the case of the "East African" trypanosoma, the stomach contents of Glossinæ were stained 3½, 6½, 23, 24, 26½ and 28 hours after a feed.

In the first film (3½ hours) the trypanosomata were fairly numerous, and showed the same changes as those described above as occurring in the later stages of the flies fed on the "Jinja" trypanosoma. The protoplasm was swollen up and vacuolated. The membrane and flagellum were unstained. The nucleus and centrosome stained well and seemed more distant from one another than usual.

In the specimens stained subsequently, these degenerative (?) changes became more marked, the protoplasm especially being more vacuolated and staining hardly at all.

In the case of the "mule" trypanosoma, more extended observations were made, and the stomach contents of Stomoxys, as well as of Glossinæ, were stained and examined. In the stomach of Glossinæ, trypanosomata of normal shape and size were seen as long as 26 and 32 hours after a feed, the nucleus and centrosome being well stained, but the protoplasm very pale. Many of the films showed curiously shaped parasites, also amœboid forms and plasmoidal masses, many vacuolated trypanosomata and some with the nucleus dividing up into smaller "chromatin" fragments. (See Plate 3.)

The stained stomach contents of Stomoxys showed trypanosomata fairly normal in appearance up to 17½ hours after a feed. In addition to these, vacuolated and deformed parasites were also seen as in the case of Glossinæ, but amœboid and plasmoidal forms were only exceptionally met with.

In a film from Stomoxys, stained 43 hours after a feed, no trypanosomata were seen, though in the fresh film made at the same time some inactive ones were present.

### (4) *Conclusions to be Drawn from the Foregoing Experiments and Observations.*

Our experiments as recorded in this Report are not sufficiently numerous and, in many cases, not sufficiently far advanced to enable us to pronounce definitively upon the nature of the four animal trypanosomata we have encountered during our researches. Certain conclusions would, however, appear to be permissible.

The disease, known locally as "Mukebi," among the transport oxen in Entebbe, associated with the presence in the peripheral blood, in the earlier stages of the disease, of the trypanosoma we have called T. III, appears to be distinct from Nagana and Surra. The trypanosoma was found to be non-pathogenic for a dog which was injected on two occasions with large numbers of the parasite. Another dog and a monkey injected with blood taken *post-mortem* from the ox, also failed to develop a trypanosomiasis, but these two latter experiments are not conclusive as demonstrating that the monkey and dog injected were insusceptible to this trypanosoma, because we failed to find the parasite on several occasions when the ox blood was examined by centrifuge during the last six weeks of the animal's life.

Further experiments are necessary to see whether this trypanosoma is pathogenic for monkeys especially, but also for other animals, for it is quite possible that the disease "Mukebi" is allied to Sleeping Sickness in man, and that the trypanosomata are identical. We endeavoured to get a fresh strain of this animal trypanosoma and, with that end in view, examined the blood of several others of the transport oxen in Entebbe, but unsuccessfully (see p. 11).

From the behaviour of the other three animal trypanosomata (T. IV, V, VI) in the stomach of Glossinæ and Stomoxys, it would seem permissible to say that these three species of trypanosoma are distinct. Unless it can be shown that very material differences in this respect in one and the same parasite can be brought about by a sojourn in, or a passage through, different mammalian hosts, the fact that T. IV was active in Glossinæ for 100 hours, T. V only 5½ hours, and T. VI for 20 hours after a feed, would lead one to infer that these three trypanosomata were distinct from one another. On the other hand, the question of their identity with any of the known trypanosomata is more difficult to answer.

The "Jinja" disease in cattle, known locally as "Sutoko," associated with the presence in the peripheral blood of the trypanosoma we have called T. IV, may be an acute form of Nagana. From the account of the disease given us by Mr. Grant, the Sub-Commissioner of Busoga, it appears that the cattle, often quite well, may be stricken down and die in 24 hours. Such a course in the case of Nagana in cattle is unusual. Then, again, this Trypanosoma produced in dogs and monkeys a more chronic form of disease than does the *Tryp. brucei;* for example, dogs lived 26, 29 and 43 days after inoculation, whereas two inoculated monkeys lived 39 and 90 days after inoculation, the former of the two dying not of trypanosomiasis alone, but of septic peritonitis. The morphology of T. IV in the blood of diseased cattle was peculiar. As mentioned on p. 18, short "stumpy" forms predominated, the average length being about 12 μ; in addition, many ordinary long forms were visible, average length about 21 μ, and in one film a very large parasite measuring 44 μ (including the flagellum) was encountered. The parasites resemble those described by Dutton and Todd in the disease amongst horses in the Gambia colony—the *Tryp. dimorphon*, whereas the largest one seen resembles the *Tryp. theileri.* (See Plate 1.)

We may sum up, therefore, and state that both morphologically and in its behaviour in naturally-diseased cattle and in artificially infected dogs and monkeys, this trypanosoma is probably distinct from the *Trypanosoma brucei.*

The "East African" disease occurring in a dog which contracted it when with the Abyssinian Boundary Commission, associated with the trypanosoma we have called T. V, may prove to be, and probably is, Surra. Morphologically, in the naturally-infected dog, this trypanosoma closely resembled the *Tryp. evansi.* (See Plate 2.)

The "Entebbe mule" disease, associated with the trypanosoma herein called T. VI, appears to be distinct from the "Jinja" disease (Sutoko), and from the East African. The trypanosoma in the mule's blood showed marked vacuolation, as it did also on several occasions in the blood of inoculated monkeys and dogs. (See Plates 2 and 3.) The parasite often had a very blunt posterior extremity. This trypanosoma, on injection, appeared to be rather more virulent for dogs and monkeys, but less so for rats, than the other two trypanosomata (T. IV and V).

In the stomach of Glossinæ it was found motile 20 hours after a feed, whereas under the same conditions, the T. IV was active 100 hours, and the T. V 5½ hours only.

### Section IV.—Is the Trypanosoma of Sleeping Sickness Conveyed by the Tsetse Flies met with in East Africa?

To settle this question one of us (E. D. W. G.) proceeded to East Africa on July 1, 1903, to organise experiments and to investigate the fly belt. Monkeys inoculated with cerebro-spinal fluid from Sleeping Sickness cases were taken from Entebbe to Nairobi, which was selected as an experimental station on account of its central position and freedom from the fly. Dr. C. A. Wiggins was associated with us in the work and after-organisation of the experiments, and continued to watch them for us until their conclusion.

On July 7 we proceeded to Kibwezi (193 miles from coast) to investigate the flies found in the "belt," and to arrange for a regular supply of living specimens being sent to the experimental station at Nairobi. Several varieties of tsetse were caught (these have been sent to Mr. Austen, of the British Museum, for investigation).*

The distribution of the fly appeared to be distinctly "local" in this district, that is to say, the flies were found in particular spots in fairly large numbers, whilst other parts were free from them. The character of the country was similar to that met with in other fly districts. The flies were said to have been very numerous about two months previously (during the rains) and many goats and cattle had died at that time. The Kikamba name for the fly is *Kitangwa*.

The flies having been caught, were fed on a healthy dog at Kibwezi and then sent by train to Nairobi. On arrival at the experimental station the flies for each experiment were fed on a healthy dog, kept for 24 hours, then placed on the infected monkey and afterwards, at the proper intervals, on the healthy monkey.

There were 4 experiments, viz.: (1) An "immediate," (2) 8 hours, (3) 24 hours, (4) 48 hours. For each experiment there was a healthy dog, an infected monkey and a healthy monkey. The object of using a dog was to be absolutely certain that the other variety of trypanosoma, *T. brucei* (to which the dog is very susceptible) was not present in the flies before putting them on the monkeys. The different varieties of flies were not separated in these experiments, as it was considered that, at the present stage, the point of most importance to settle was, whether *any* of the species met with in East Africa conveyed the trypanosoma of Sleeping Sickness.

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* Mr. Austen tells me that up to the present no *Gl. palpalis* have been sent to him from East Africa. The flies sent are *Gl. pallidipes*, *Gl. fusca*, and *Gl. longipennis*.—D. N.

The same experiments might be carried out with each individual species. The details of the two positive experiments are given below. It will be seen from these that the tsetse flies met with in East Africa are capable of conveying the trypanosoma of Sleeping Sickness after 8 hours and 24 hours. This is a point of considerable importance, as the belt extends down as far as South Africa and the fly also runs up the great waterways from the coast. Hence should this belt become infected by the trypanosoma of Sleeping Sickness, the distribution of this disease would be very greatly extended.

### EXPERIMENT 156.—Kikuyu Monkey. "8-hour" Experiment.

July 17, 1903. Feeding started.
August 15. Tryp. absent. Mal. present.
August 22. Tryp. absent. Mal. present.
October 1. *Tryp. present.*

The duration of this experiment was about two and a half months, and an average of four or five flies a day fed on this monkey.

### EXPERIMENT 157.—Kikuyu Monkey. "24-hour" Experiment.

July 10, 1903. Feeding started.
August 15. Tryp. absent. Mal. absent.
August 22. Tryp. absent. Mal. absent.
September 8. Experiment interrupted, as all flies were killed by ants.
October 4. Feeding resumed.
October 25. Blood examined. Tryp. present.
November 4. Monkey brought to Entebbe.
November 9. Monkey died.

*Post-mortem.*—The organs were all found to be healthy.

Death was due probably to the change of climate.

On an average four flies a day fed on this monkey.

## Section V.—FURTHER OBSERVATIONS ON THE DISTRIBUTION OF THE TSETSE FLY IN UGANDA.

After Col. Bruce's departure we made observations in special areas which had not been previously investigated.

(1) The Busoga (right) bank of the Nile beyond Kakoge ferry. This was searched for a distance of about 20 miles, in Gabula's country, but no tsetse flies were found.

(2) The Nile beyond Lake Albert. The Collector at Wadelai, Mr. W. Y. Wyndham, sent us some *Glossina palpalis* from the bank of the Nile on the Congo side. He was then instructed by H.M. Commissioner to examine the shore of the Lake Albert, and on October 25

the following telegram was received from him: "Just finished tour of Lake Albert, have found tsetse fly prevalent everywhere along coast; details follow by post."

In his letter Mr. Wyndham states that at each of 12 places situated on the shores of the lake (six on the Uganda shore and six on the Congo Free State shore) *Glossina palpalis* was found, and he forwarded specimens of this fly to us from each place. One paragraph in his letter is particularly interesting, so we give it in full. "Mahagi Kabir was interesting, as I found the fly on the banks of a river of some size, unmarked on the maps, called the Kakoi. There was neither forest nor swamp, but the banks were covered with high elephant grass. The fly cannot depend for its existence upon game, as in most of the places in which I found it there was none, or next to none."

Although the *Glossina palpalis* was present all round the Albert Lake, there was, at that time at any rate, no Sleeping Sickness in the district.

(3) The left bank of the Nile, between the Lakes Victoria and Choga, was investigated, including the districts known as Bugerere and Buruli, but no tsetse flies were found.

(4) One *Glossina palpalis* was received from the "White Fathers," said to have been caught near the mission station at Fort Portal, in Toro. As we had hitherto not received any tsetse flies from that district, we asked for more specimens to be caught and sent in, if possible.

## *Conclusion.*

In conclusion, we beg to offer our hearty thanks to Colonel Hayes Sadler, C.B., His Majesty's Commissioner and Consul-General for Uganda, for the assistance he has invariably given us: to Drs. Moffat, C.M.G., Hodges, Wiggins and Ross for their active co-operation in the work of the Commission; to Mr. R. J. Stordy, the Principal Veterinary Officer to the Protectorates, for sending us the dog suffering from the "East African" disease and also for taking the photographs of Sleeping Sickness patients; to Mr. W. Grant, C.M.G., Sub-commissioner for Busoga, for much practical help and information concerning the "Jinja" disease in cattle; to the Sub-commissioners, collectors and other officials of the protectorate, to the bishops and missionaries of the various churches, and to the Prime Minister and Regents of Uganda, for their co-operation in collecting flies for identification; and, lastly, to Dr. A. C. Stevenson, of University College, London, for the sketches illustrating the different forms of trypanosomata described in this Report.

## DESCRIPTION OF PLATES.

### PLATE 1.

FIG. 1.—Film of "Jinja" animal, showing trypanosomata of varying sizes and forms (T. IV). × about 1200.

FIG. 2.—*a.* Filaria from blood of Monkey 241, showing general appearance. × 500.

*b.* Head end. *c.* Break in protoplasm.

*d.* Tail end. *b, c, d.* × 1200.

### PLATE 2.

*a.* Trypanosomata in blood of Dog 160 (T. V).

*b, c.* Trypanosomata in blood of Dog 197 (T. VI). (All × about 1200).

The five parasites in the lower half of this plate represent vacuolated and deformed trypanosomata (T. VI) in blood of Monkey 180. × about 1200.

### PLATE 3.

Various forms of trypanosomata seen in a film of stomach contents of *Glossina palpalis*, 14½ hours after feeding—"Mule," T. VI. × 1200.

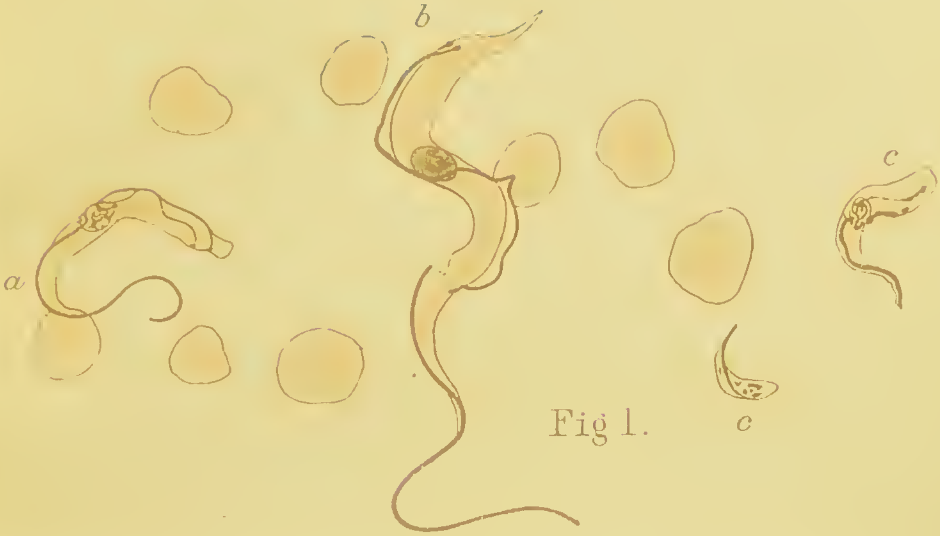


Fig 1.

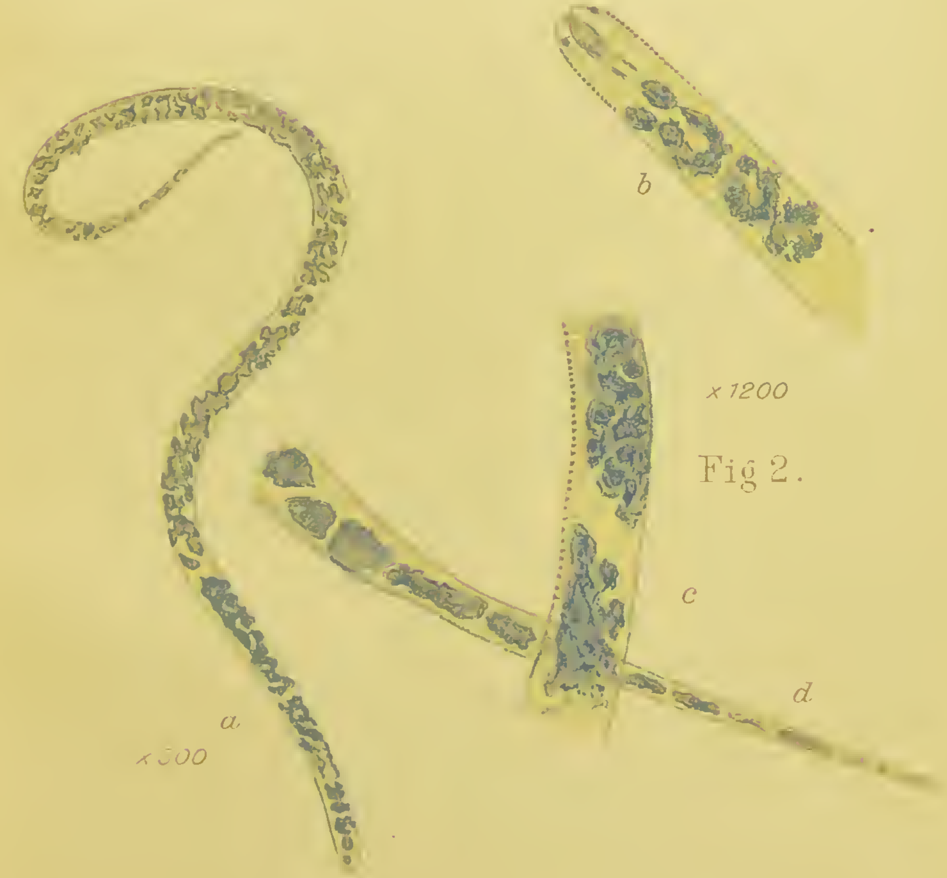
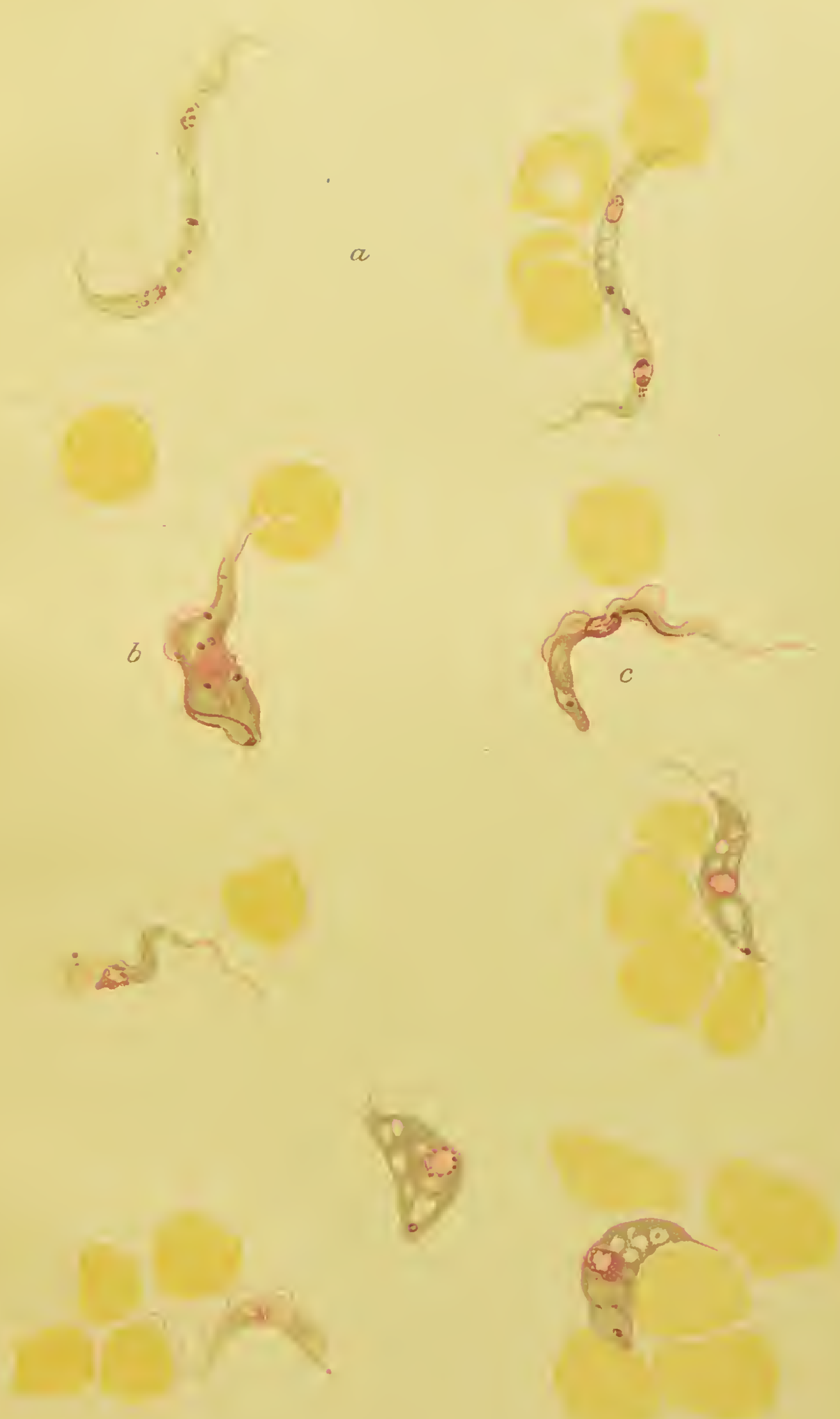


Fig 2.

*Greig & Nabarro* *Sleeping Sickness Report Pl. 2.*



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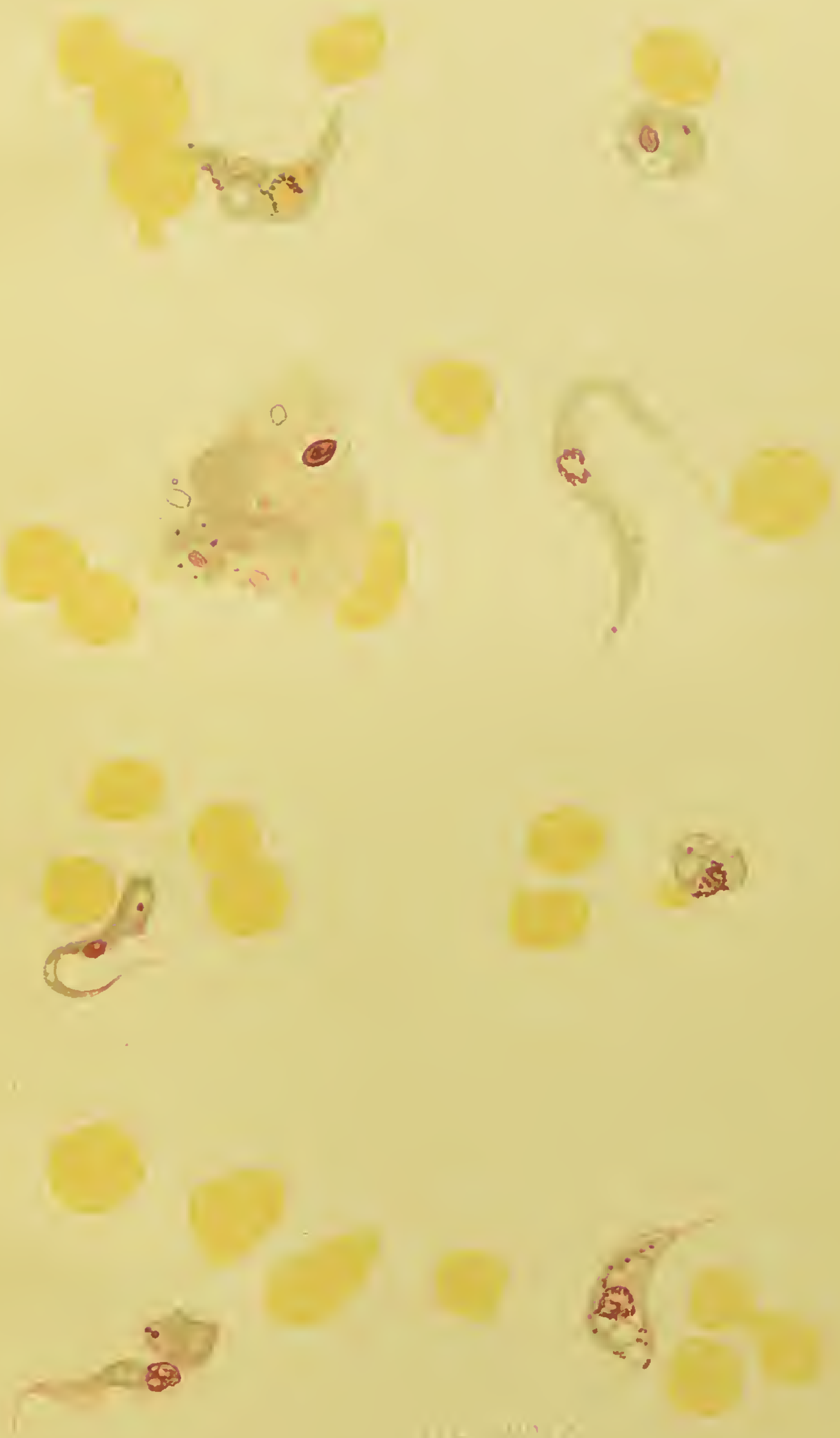

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